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SOME OBSERVATIONS ON PHONOCARDIOGRAPHY.

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INTRODUCTION.

THE desire to see graphic records of the heart sounds was a natural enough corollary to the improvement that occurred in the stethoscope, from Laënnec's time (1816) to the end of the nineteenth century. During this period binaural stethoscopes with non-rigid tubing and bell chest-pieces were introduced. In 1901 the diaphragm chest-piece was patented by Bowles. After this further improvement of the stethoscope appeared unlikely.

Einthoven in 1907 made the first attempt at recording heart sounds, using a carbon-granule microphone and a galvanometer. He was successful to a degree and his pioneer work was rapidly followed by many other experimenters.

In 1924 the audion tube amplifier became a practical reality, and the Western Electric Company developed one of the first practical amplifying stethoscopes.

With advances rapidly occurring in the radio field, new and better machines were being produced and so today most machines embody the latest developments in the science of acoustics.

HEART SOUNDS AND THEIR RECORDING.

The Frequency Range and Sound Characteristics of Heart Sounds.

Practically all the sounds of interest in auscultation are made up of frequencies below 1000 cycles per second.

Presystolic murmurs as a class are characterized by a greater predominance of low frequencies than other murmurs and are almost invariably termed low pitched, in contrast to the aortic diastolic murmur that is characteristically high pitched.

The frequency bands of importance in systolic and diastolic murmurs are broadly the same. In each group extremely low-pitched and high-pitched sounds are found, but there appears to be nothing in the quality of the sounds that is characteristic of murmurs occurring in systole or diastole.

Williams and Dodge (1926) observed the distribution of energy in different frequencies of the normal heart sounds. They noted the high percentage of energy in the low-frequency range.

Moreover, they found that "low-pitched" heart murmurs are composed of frequency components below 400 cycles per second and that "high-pitched" murmurs range from 120 to 660 cycles per second.

Rappaport and Sprague (1941) have not encountered any heart sounds or murmurs of any noticeable value above 650 cycles per second, but have encountered some frequency components of heart sounds or murmurs at five to ten cycles per second that were well below the lower range limit of the human ear (20 cycles per second).

Factors Modifying Heart Sounds.

Modification by Thoracic Contents and Chest-Piece.

A large part of the sound energy of the heart never reaches the surface because of losses due to viscosity, elasticity *et cetera*. The sound energy is further modified by the type of chest-piece, the stethoscope tubing and the human ear.

Contrary to older opinion, the open bell should not be considered primarily as an accumulator. When an open bell is applied to the chest the skin forms a diaphragm

and the subcutaneous tissues under it act as a damper. The net result is what is described technically as a damped diaphragm; the maximal sensitivity of this is at the frequency corresponding to the natural period of vibration of the diaphragm, which in turn is dependent on its inherent inertia, elasticity, diameter and tautness. The practical application of this is in altering the pressure of the bell-piece to procure high-pitched or low-pitched sounds—for example, light pressure will bring out third heart sounds and gallop rhythms, and firm pressure is helpful in the hearing of high-pitched diastolic murmurs, as at the aortic area.

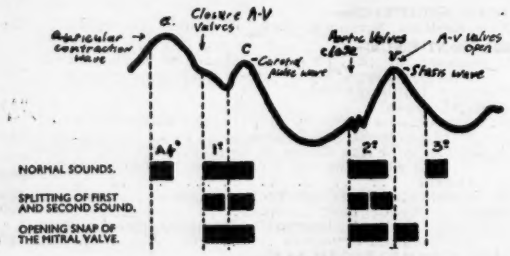

FIGURE I.

Heart sounds and venous pulse (after Orias and Braun-Menendez). Note the relations between the venous pulse and the diastolic sounds. The second component of the split second sound appears before the summit of the *v* wave; the opening snap of the mitral valve coincides with the summit of the *v* wave; the physiological third sound coincides with the last portion of the descending limb of the *v* wave, and the auricular sound coincides with the *a* wave and is the site of the fourth heart sound.

This was proved by the work of Rappaport and Sprague (1941), who used different sized stethoscope bells in a series of experiments. They found that the larger the diameter of the open chest-piece, the better the response to low-pitched sounds and *vice versa*. The smaller the bell and the more firmly it is placed on the precordium, the more attenuated become the low-pitched sounds and the louder the higher frequency components. The diaphragm type of chest-piece (Bowles type) when applied to the chest wall is similar to that of the small open bell in that attenuation of low-pitched sounds is obtainable. This may be an advantage, for example, in unmasking a higher pitched aortic diastolic murmur. It may be, on the other hand, a disadvantage and fail, for example, to detect the low-pitched diastolic rumble of mitral stenosis.

Realization of these factors is an important technical requisite for correct phonocardiographic procedure. Several times we were struck by the noticeable difficulty of recording the diastolic murmur at the mitral area of rheumatic valvular disease, particularly in view of the work of Evans (1947), who states that in each of his 74 cases of mitral disease with systolic murmurs beginning at the *S* line or before, there was in every case a mid-diastolic murmur "to give proof of mitral stenosis". We later came to realize that it is quite possible for the technician so to attenuate this murmur by an incorrect chest-piece (for example, by using a diaphragm or too small a bell) or by too firm a pressure (caused by tightening the elastic holding-strap) that the murmur is no longer registrable. The time has indeed come when to get a reasonably accurate record we must adopt the optimum chest-piece, adjust the pressure to correct tension and, policy of perfection, take tracings at differing frequency bands.

TABLE I.
First Heart Sound.

Components.	Logarithmic Registration.	Linear Registration.	Stethoscopic Registration.	Types of Vibration.
First ..	None.	Good.	Attenuated.	Low frequency. Coarse and intense.
Second ..	Good.	Good.	Good.	High frequency.
Third ..	Good.	Good.	Good.	High frequency.
Fourth ..	None.	Good.	Attenuated.	Low frequency. Coarse and intense.

Mannheimer (1940) has already developed a phonocardiograph in which the heart sounds are split into six frequency bands.

With regard to the latter, Nylin and Biörck (1947) have demonstrated a murmur of auricular origin in a case of mitral stenosis and heart block. They have objectively proved that this murmur shows its greatest graphic response on a frequency band of 15 to 100 cycles per second and virtually no response on a 400 to 850 cycle range, a finding which indicates the importance of bearing in mind the necessity of "scanning the frequency bands" or of having a machine that faithfully records all the necessary range in an accurate manner.

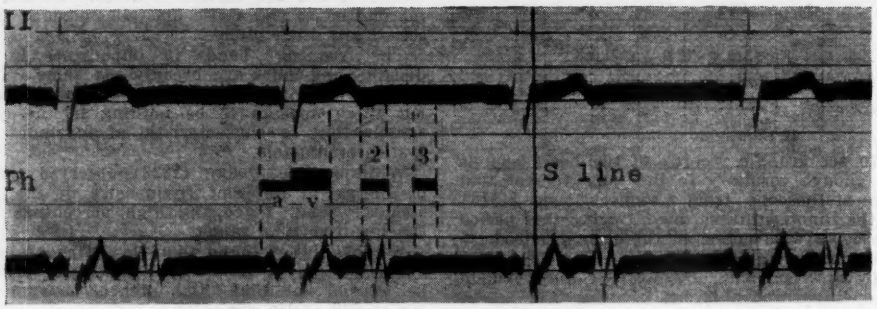

FIGURE II.

Normal phonocardiogram (from William Evans, 1947) showing the heart sounds in relation to the electrocardiogram. The *S* line drawn at the end of the *S* wave of the electrocardiogram, and continued to intersect the phonocardiogram, marks the early phase of ventricular contraction. The auricular (*a*) and the ventricular (*v*) moieties of the first heart sound are shown, as well as the second (2) and third (3) heart sounds.

Modification by Rubber Tubing of Stethoscope.

Rubber tubing also has an effect in modifying the heart sounds further. Rappaport and Sprague (1941) state that with sounds "below 100 cycles per second the efficiency is not affected materially by tubing length". From 100 to 1000 cycles per second tubing length exerts a considerable

effect on efficiency. For example, at 200 cycles per second there is a 15-decibel attenuation of the sounds when two rubber tubes each 25 inches long are substituted for two similar tubes each three inches in length. The sound would be eight times as loud in the short-tubing system. This attenuation occurs in the region in which the low-intensity, high-pitched, aortic diastolic murmurs are present, and every possible measure in efficiency in this region is of the utmost value.

Modification by the Human Ear.

Normal hearing deteriorates with age, and it does so in the form of an increase in the "valleys" of the "peak and valley" pattern which is normal for the auditory range, so that certain sounds gradually become inaudible to the listener without his awareness of it. Hence one of the values of a graphic recording system.

On Graphic Registration of Heart Sounds.

There are three distinct types of phonocardiac registrations: (i) linear, (ii) stethoscopic, (iii) logarithmic.

Linear Registration.

The linear registration is a representation of sounds as they occur at the surface of the chest wall—that is, the deviation of the tracing is proportional to the intensity of the vibration at the surface of the chest. Since large-intensity, low-pitched vibrations may be 10,000 times as great as the minimum recordable high-pitched sounds, to make the tracing practical the latter are not recorded when the sensitivity is reduced to keep the big amplitudes on the tracing.

Orias and Braun-Menendez (1939) have produced tracings of the heart sounds by a simple mechanical method giving linear reproduction of great clarity and accuracy within a limited range. However, their technique is applicable only as a laboratory method. True linear phonocardiography can be obtained electrically throughout all frequency bands only by using machines something like those of Mannheimer or Nylin and Biörck, in which the sound range is split up into many frequency bands. Whether use of such machines will add much to clinical cardiology remains to be seen.

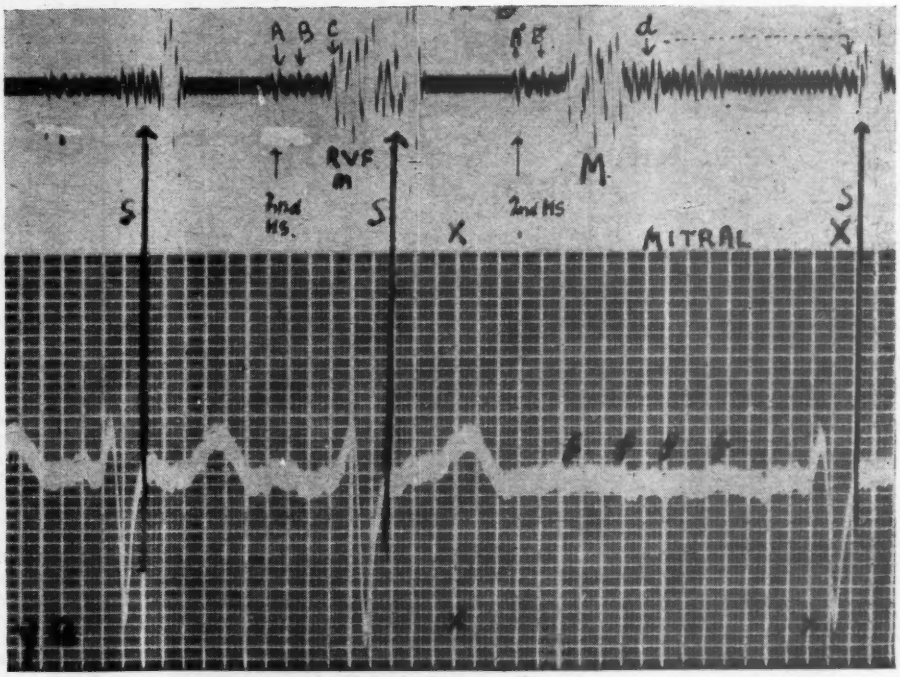

FIGURE III. Mitral stenosis and auricular fibrillation. See text.

The human ear registers sound vibrations in a "non-linear logarithmic manner". This means that the human is able to perceive a high-frequency note much more easily than he can the lower register, as can be seen from the audiogram. It requires, for example, in dynes per square centimetre of drumhead, 10,000 times as much pressure at 20 cycles per second to produce a just perceptible sound as it does at 512 cycles per second.

A phonocardiographic instrument registering sounds as interpreted by the human ear is called "logarithmic" and is quite distinct from the "stethoscopic" phonocardiograph which registers sounds as they are presented to the ear by the stethoscope. This clarification is really important, because if the phonocardiograph has only the characteristics of the human ear and is therefore logarithmic, it will record only what the ear hears. If on the other hand it is a true stethoscopic phonocardiograph, it will record some sounds not heard by the human ear but of clinical importance. Thus the ear hears only the second and third components of the first heart sound, but harmonic and frequency analysis shows that there are four components. The stethoscopic phonocardiograph can often pick up the other components as well.

Stethoscopic Registration.

The stethoscopic system of registration gives a representation of how sounds are presented to the ear drums after modification by chest-pieces and by the characteristics of the instrument itself. The characteristics of the stethoscopic phonocardiogram in use on our own records are discussed later.

Logarithmic Registration

The logarithmic system of registration records heart sound vibrations, not as they occur, but as they are perceived by the human brain when an average stethoscope is employed—that is, the sounds are recorded in proportion to their relative loudness. Loudness is a

subjective sensation composed of intensity and pitch, and an attempt to record this "loudness" requires a distortion of the true linear record by introducing the "peculiar logarithmic response of the human ear" as exhibited on an audiogram. Such a tracing, faithfully reproduced, would show just what the brain perceives, no more and no less. Thus we hear only the second and third components of the first heart sound and the second component of the second heart sound, and such a tracing would show only these; it would omit the first and fourth components of the first heart sound and the first, third and fourth components of the second heart sound.

On Reference Tracing.

Most workers use the electrocardiogram prepared simultaneously as a reference line for heart sounds, but Orias and Braun-Menendez (1939) prove that the electrocardiogram as a reference tracing is unsatisfactory, for apart from the beginning of the *P* wave and of ventricular excitation (the beginning of the *QRS* complex), it does not give any other reference point that can be justifiably connected with the mechanical activities of the heart. The electrical manifestations of the heart's activity are not synchronous with its mechanical activity. These authors point out that it is impossible to recognize on the electrocardiogram with absolute certainty any of the phases of cardiac activity, in either systole or diastole. To analyse a phonocardiogram it is not enough merely to know when the electrical changes associated with systole begin and end. The only accurate reference for sound tracings is the venous pulse taken from the jugular vein by a simple phlebogram (see Figure I). This reflects the mechanical activity which gives rise to sounds. Be it as it may, the time may come when all phonocardiograms will include the phlebogram. It remains to be seen whether much of the work that has been done with the electrocardiogram as a reference tracing will become invalid or whether further use will confirm its practical value despite admitted theoretical objections. This point is discussed later.

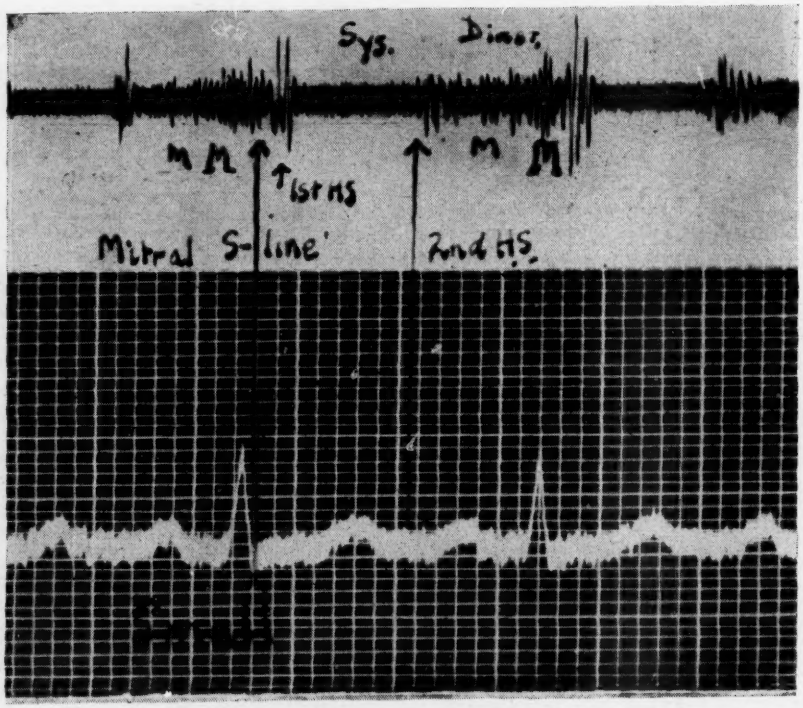

FIGURE IV. Mitral stenosis.

Composition of Heart Sounds.

First Heart Sound.

Caeiro and Orias (1937) have described the first heart sound as being composed of four components: (i) residual vibrations of auricular origin; (ii) vibrations produced by the beginning of isometric contraction in the cardiac cycle, *plus* closure of the mitral and tricuspid valves; (iii) opening of the semilunar valves; (iv) the vibrations from the acceleration of blood into arteries during the maximum ejection phase of ventricular systole.

In the following table (Table I) the registration of these four components by logarithmic, linear and stethoscopic phonocardiography is shown.

The length of the first heart sound is limited by the *C* wave of the phlebogram (Figure I). Vibrations after this are due to a systolic murmur.

The onset of the first components of the first heart sound should be before the apex of the *QRS* complex in the electrocardiogram. William Evans (1947) uses the *S* line of the electrocardiogram as a reference for the interpretation of systolic murmurs (see Figure II). It would correspond roughly to the onset of the isometric phase (ii) mentioned above and is discussed later.

Second Heart Sound.

The second heart sound also has four components: (i) vibrations due to the beginning of the diastolic fall in pressure with ventricular wall relaxation; (ii) closure of the semilunar valves (termination of ventricular systole); (iii) vibrations of arterial walls and blood columns; (iv) opening of the mitral and tricuspid valves.

The phonocardiograph may register all four components, but the human ear picks up only one, that is, the second, and hence the second heart sound appears shorter than the first.

The last component is inaudible and, like the first and third components, does not show on logarithmic phonocardiograms, but it does on linear and stethoscopic cardiograms. This component is audible occasionally in mitral stenosis and is described as the opening snap of the mitral valve.

The second heart sound continues no longer than the apex of the *v* wave in the venous tracing (Figure I). In the electrocardiogram the second heart sound roughly corresponds to the end of the *T* wave.

Third Heart Sound.

It is certain that the third heart sound is due to vibration of the ventricular walls, including valvular structures, set up by the sudden inrush of blood in initial moments of diastole immediately after the opening of the auriculoventricular valves. Best heard between inspiration and expiration, it is not heard in every cycle, but it is slightly increased in intensity and raised in pitch by pressure on

the abdomen. The time of onset is in front of the lowest portion of the descending limb of the *v* wave of the venous tracing (Figure I). It is often heard in normal hearts and follows the second heart sound by about 0·08 second; it lasts for 0·04 second. It is heard at the apex in many normal young adults. It is intensified by procedures

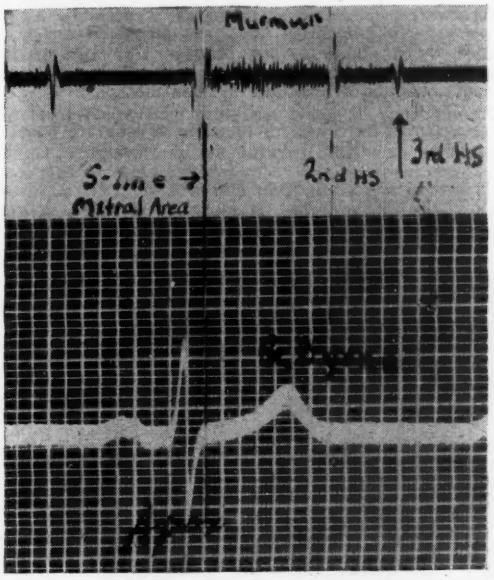

FIGURE V. Triple rhythm.

which increase the venous flow into the auricles—for example, recumbency or exercise.

The third heart sound has come considerably into prominence with the rebirth of phonocardiography, and the whole subject of triple rhythm is undergoing reassessment.

CLINICAL APPLICATION.

The following records were obtained with a Cambridge instrument, a bell-shaped chest-piece being used and fixed to areas over the precordium with an elastic strap passing around the thorax.

In the light of further inquiry many of these records could be improved by attention to the detail of the acoustic chest-piece, pressure on the skin and the type of murmur sought.

Although born about the same time as electrocardiography, phonocardiography has languished until recent years. As William Evans (1948) pointed out at a recent international conference of physicians, it shows signs of a considerable revival. There seems to have developed in modern cardiology an imperative need for the registration of heart sounds. Without deprecating in any way the value of clinical auscultation, it must be conceded that the human ear is unable to differentiate in many cases the various sound effects that take place in systole and diastole. Also because of the peculiar logarithmic response of the human ear that we have already described, many sound effects are entirely inaudible or masked that are clearly registrable on a phonocardiogram.

The French school headed by Lian and Minot believe that the main value of sound records is in establishing the presence of triple rhythm. They state that sound records enable the triple rhythm of left ventricular failure to be differentiated from the unimportant reduplication of the first sound, and the third heart sound of mitral stenosis from reduplication of the second sound. The French workers seem to regard phonocardiography as a means of recording what can actually be heard and thus differ from the British and American schools.

Triple Rhythm.

Triple rhythm occurs whenever there is an interpolated sound and is in contrast to dual or quadruple rhythm. Gallop rhythm is due to the peculiarity of cadence and tachycardia likening certain types of triple rhythm to the gallop of a horse.

The term gallop has become rooted in the history of triple heart sounds, but to prevent misunderstanding it is better to discard it or to remember that the word gallop is limited to a peculiar cadence and tachycardia. Exactly the same significance may be attached to a triple rhythm lacking the peculiar "gallop" cadence occurring, for example, with slower heart rates.

Crighton Bramwell (1935) insists that the word gallop be restricted to a presystolic sound occurring most frequently in patients with hypertension, advanced coronary disease or acute inflammatory lesions of the heart.

Evans (1943) advocates adoption of the term triple rhythm and discarding of the term gallop. This author then proceeds to classify triple rhythm into three types. Type I*a* is due to addition of the third heart sound in health and Type I*b* to its addition in right ventricular failure. Type II is due to addition of the "fourth heart sound", Type II*a* to delayed auriculo-ventricular conduction and Type II*b* to left ventricular failure. Type III is due to an extra sound in late systole and is unimportant.

Orias and Braun-Menendez (1939) simplify classification of gallop rhythm into (*a*) rapid filling gallops, when the sound coincides with the end of the rapid ventricular filling phase; (*b*) presystolic (or auricular) gallops, when the extra sound coincides with presystolic or auricular systole; and (*c*) complete or incomplete summation gallops, according to whether the sounds due to auricular con-

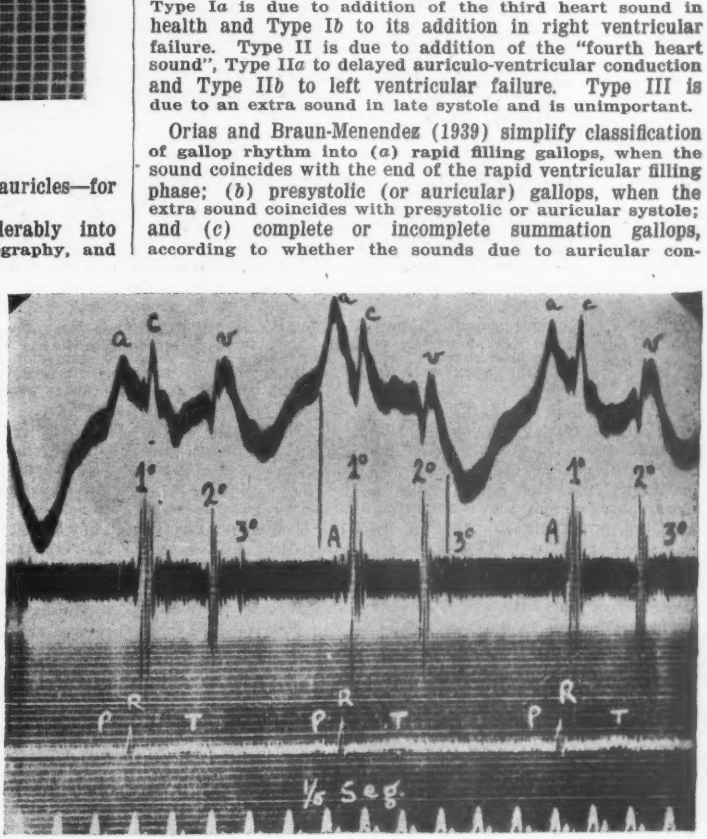

FIGURE VI.

Third heart sound in pregnancy (Pereira from Orias and Braun-Menendez). The phonocardiogram shows a physiological auricular sound (A) and a physiological third heart sound (3°) occurring at the time of the descending limb of the *v* wave.

traction and rapid ventricular filling, which appear simultaneously, do or do not coincide exactly.

The general clinical feeling about triple rhythm is that its presence contributes little to our knowledge of the state of the patient. It is felt that many triple rhythms have the cadence of a true gallop and yet are innocent, and that the significance of triple rhythm cannot be determined without reference to the clinical condition of the patient.

Does phonocardiography enable us to distinguish the sinister from the innocent, and have we any justification for assuming that there are different types?

Evans would say that his Type I*a* is the only innocent type and that he can distinguish it from the other types by its site, the effect of posture and its time of onset in the phonocardiogram. It is this last criterion that we feel may be fallacious, since sounds do not all arise in their expected positions on the tracing. For example, he identifies an interpolated sound occurring during the *P-R* interval as due to auricular systole, and calls it triple rhythm due to a fourth heart sound. Orias and Braun-Menendez, on the other hand, constantly stress the error of identifying the origin of a sound by means of the electrocardiograph alone. It is an error to assume that a sound occurring in the *P-R* interval is always due to auricular systole. This is well shown in one of the tracings in a later section of this article, in which the sound is produced by rapid ventricular filling in the presence of fibrillation and absence therefore of auricular contraction, and in a period immediately preceding systole (Figure III).

Evans maintains that his triple rhythm Type II can change to Type I with clinical improvement. It remains to be seen with further phonocardiography whether there is a Type II*b* auricular triple rhythm or merely Type I, a sound due to the rapid ventricular filling phase occupying respectively the presystolic or mid-diastolic period of diastole.

Until these points are cleared up with further experience and adequate phonocardiography we feel that the phonocardiograph at present offers no help to clinical medicine in the elucidation of triple rhythm.

Heart Murmurs.

The problem of phonocardiography in regard to murmurs is not so much the confirmation of the significance of murmurs about which nobody is in any doubt, as, firstly, the elucidation of those murmurs the significance of which is doubtful and, secondly, the detection and elucidation of inaudible or masked murmurs. No class of murmur, for example, has been hitherto more contentious than the isolated apical systolic murmur in a presumably healthy young adult.

Here we must turn to Evans again, who states that "the significance or otherwise of a systolic murmur may be decided by a sound record because its exact position, which is more important than either its quality or intensity, can be determined by this method".

Evans's work on heart murmurs endeavours to simplify the location of murmurs in relation to separate phases of the cardiac cycle, by drawing a line through a point on the electrocardiographic reference tracing at the end of the *S* wave. A murmur that precedes the *S* line, he states, is the effect of auricular systole. If it starts at the *S* line it coincides with the early phase of ventricular systole; if it starts a little later than the *S* line the murmur occupies mid-systole. Next the customary form of the graphic complex depicting the separate normal heart sounds should be studied in order that the variant produced by the addition of a murmur may be recognized. They differ one from the other only in the frequency of the vibration which creates them. The oscillations produced by a heart sound are fewer or coarser; those produced by a murmur are more numerous and finer.

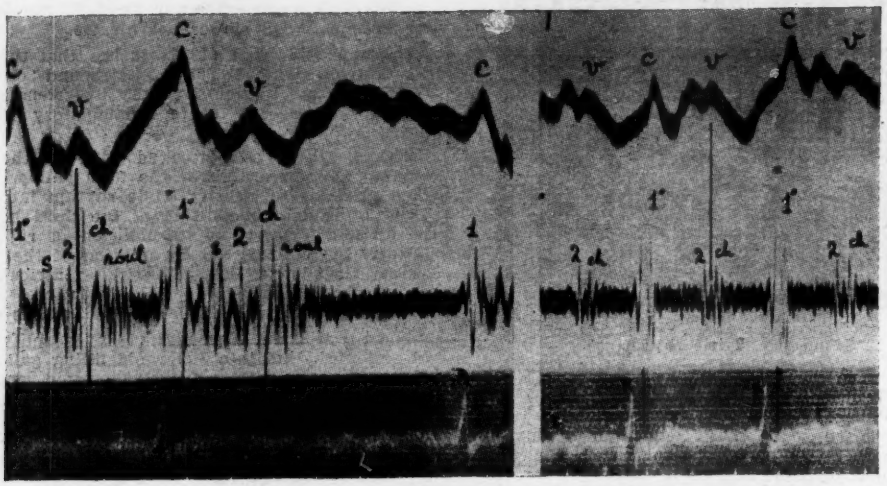

FIGURE VII.

Heart sounds in mitral stenosis (Battro and Braun-Menendez, 1937). The phonocardiogram on the left shows an opening snap (Ch) followed immediately by the diastolic rumble (roul). The phonocardiogram on the right shows the opening snap alone. The former tracing was recorded at the apex area and the latter at the mesocardiac area. Note the time relationship of the snap to the apex of the *v* wave.

Among many points arising out of this investigation in regard to the location of murmurs we should like to draw attention to three points particularly: (*a*) the identification of the innocent mitral systolic murmur; (*b*) the auricular origin of the rheumatic mitral systolic and presystolic murmurs; (*c*) the allegedly almost invariable accompaniment of a mid-diastolic murmur as well as a presystolic murmur in mitral valvular disease.

Despite the criteria mentioned above we often found difficulty in identifying the beginning of a murmur and in distinguishing heart sound from murmur. Because of this we were often left in the same equivocal position at the end as we were before phonocardiography in regard to the innocence or otherwise of the murmur, particularly if the intensity of the murmur was moderate to slight.

The second point has already been raised in the previous section on triple rhythm. It is that, although the phonocardiograph record preceding the *S* line is probably

approximately related to presystole, a sound or murmur occurring therein is not necessarily auricular in origin, although it often is.

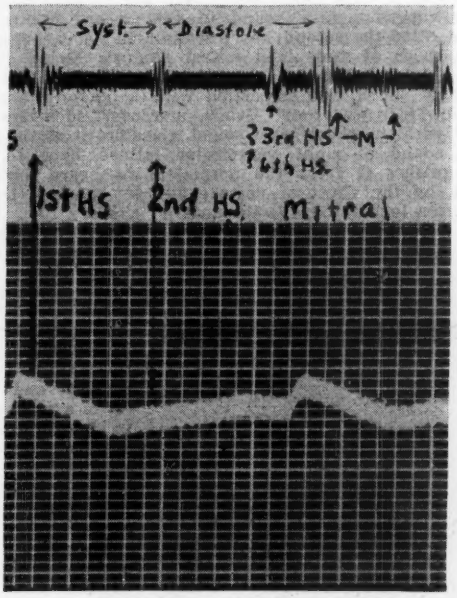

FIGURE VIII. Triple rhythm. Congestive heart failure.

This point we hope to show in several of our tracings, one with mitral stenosis and fibrillation in which the presystolic murmur could have nothing to do with auricular systole.

The third point we have raised we found to be the most contentious of all. Far from being almost invariable, we found the diastolic murmur to be not infrequently completely absent in mitral valvular disease. Moreover, we feel that it is completely erroneous to speak of it as the mid-diastolic murmur of mitral stenosis. This point is raised again later in our tracings. The position it occupies in diastole depends entirely on the position in the cardiac cycle occupied by rapid ventricular filling.

ILLUSTRATIVE TRACINGS.

Auricular Fibrillation and Mitral Stenosis.

Figure III.

C.B., a female patient, aged thirty years, has a history of "growing pains" as a child and was treated in hospital two years ago for rheumatic fever. On examination her heart is enlarged; what is thought to be a presystolic and diastolic murmur is audible at the mitral area with a loud first sound. The blood pressure is 130 millimetres of mercury (systolic) and 60 millimetres (diastolic). The pulse is fibrillating.

Comment.—The phonocardiogram recorded at the mitral area is very interesting, as during a period of non-conduction of auricular impulses the sound track shows activity that must be ascribed to the auricular blood passing through a stenosed mitral valve.

The concept of auricular contraction as the genesis of the presystolic murmur of mitral disease is implied in the statement that the murmur disappears in fibrillation. We are also told that in advanced cases of mitral disease with fibrillation a diastolic murmur is alone heard without the presystolic accentuation. This diastolic murmur invariably begins in mid-diastole according to Evans, and never earlier, whilst Lewis maintains that with slow fibrillation and stenosis the "favoured part is early diastole". We should like to suggest that these statements are either totally or partially false. In the first place the diastolic murmur in stenosis is due to the rapid filling of the left ventricle. This phase of diastole occurs after the opening of the auriculo-ventricular valves and is identified on the phlebogram as coinciding with the descent of the *v* wave, where the apex of the *v* represents the opening of the auriculo-ventricular valves.

At normal rates the rapid ventricular filling phase occurs from the early to the mid-part of diastole. Tachycardia shortens the diastolic part of the heart cycle, causing the phase of rapid ventricular filling to occur simultaneously with the phase of auricular contraction. This may cause the murmur due to rapid ventricular filling to occur immediately before the *QRS* complex of the electrocardiogram, and hence it appears presystolic in time despite the presence of fibrillation. This is well illustrated in one cycle (heart rate 120 per minute) depicted in Figure III, in which such a presystolic murmur (R.V.F.M.), due to rapid ventricular filling, occurs. Evidence that it is not of ventricular origin is seen in the next cycle, when during a period of ventricular standstill a similar murmur follows in the early diastole and must be ascribed to rapid ventricular filling through a stenosed mitral valve. In the absence of ventricular contraction the blood continues to flow into the ventricle and the murmur is considerably prolonged.

The final point to consider in this phonocardiogram is the identification of A and B. It is highly likely that A (A′) is the second heart sound, and it is highly likely that B (B′) is the opening snap of the mitral valve occurring 0·08 second after the second heart sound. This could be told only by examination of a simultaneous phlebogram; if B was the opening snap of the mitral valve it would correspond to the apex of the *v* wave and C to the downstroke of the same wave.

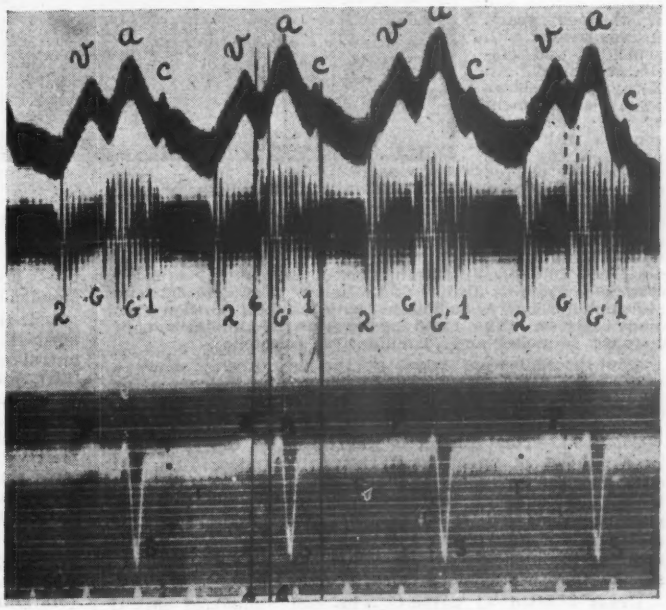

FIGURE IX.

Incomplete summation gallop. Mitral tracing (Orias and Braun-Menendez). The extra sound has two definite components (G and G′) very near one another and corresponding respectively to the end of rapid ventricular filling and to auricular systole.

Rheumatic Valvular Disease: Mitral Stenosis.

Figure IV.

J.S., a female, aged fifty-five years, has a history of rheumatic fever on three or four occasions during childhood, the last occasion being at the age of twenty-one years. She has had three children without difficulty, the confinements being normal. An embolic cerebral episode occurred one year ago. On examination of the patient, the blood pressure is 150 millimetres of mercury (systolic) and 100 millimetres (diastolic). The apex beat is in the fifth intercostal space four inches from the mid-line. A pronounced presystolic murmur is audible at the apex accompanied by a thrill and diastolic rumble. Fluoroscopy and X-ray films show much left auricular enlargement.

Figure IV shows a murmur commencing about mid-diastole with a marked presystolic accentuation and an accentuated first sound. It also illustrates the difficulty of relating the second heart sound to the exact point of the end of the *T* wave as recommended by Evans.

Comment. — This phonocardiogram is shown to illustrate the "classical" features of rheumatic valvular disease and well demonstrates the features described by Evans of this type of lesion.

Triple Rhythm.

Figure V.

G.B., a male, aged fourteen years, suffered from rheumatic fever at the age of seven years and from subacute bacterial endocarditis at the age of thirteen years. He was treated with penicillin and discharged cured. An X-ray film shows some enlargement of the pulmonary conus with the patient in the right anterior oblique position and fluoroscopy reveals also some visible enlargement of the right and left auricles. On clinical examination of the patient a harsh systolic murmur is audible at the mitral area. No diastolic murmur or sound is audible in the left lateral position even after exercise. The blood pressure is 120 millimetres of mercury (systolic) and 70 millimetres (diastolic).

A phonocardiogram taken at the mitral area shows a systolic murmur commencing at the *S* line and extending through systole. A distinct supernumerary sound is recorded towards mid-diastole, but no diastolic murmur is detected.

Comment.—This tracing is interesting in that it occurs in a subject of unequivocal rheumatic endocarditis. It could equally well occur in other patients whose rheumatic history is doubtful. How far does such a phonocardiogram confirm that the recorded murmur is an organic murmur? We are presented with a tracing of a systolic murmur commencing at the *S* line and a supernumerary sound constituting one form of triple rhythm.

Evans studied 41 patients who had undoubted mitral stenosis with systolic murmurs, but in whom no presystolic murmurs could be heard clinically. He found that in 33 cases the murmur commenced in the *P-R* interval of the electrocardiogram and in eight it commenced at the *S* line. In every one of his 41 cases a diastolic murmur was present as well. He further regards the association of this type of systolic murmur and a mid-diastolic murmur as pathognomonic of rheumatic mitral disease. In the present case there is a typical systolic but no diastolic murmur.

The supernumerary sound in this record occurs 0·12 second after the second heart sound. In an earlier study of 70 cases of undoubted mitral stenosis Evans (1943) found triple rhythm in 44 cases. In 30 of these 44 cases the third sound was associated with a diastolic murmur and in 14 it was not. Evans, moreover, identifies this sound with the third heart sound, classifies it as the third heart sound of right ventricular failure Type I*b*, and distinguishes it from the normal third heart sound of health by the effect of posture and by the fact that it was often louder towards the anterior axillary line. Orias and Braun-Menendes, on the other hand, state that the electrocardiogram is "totally inadequate for the identification of the third heart sound" and that "it is a gross error to identify diastolic sounds by the interval occurring between them and preceding second sound".

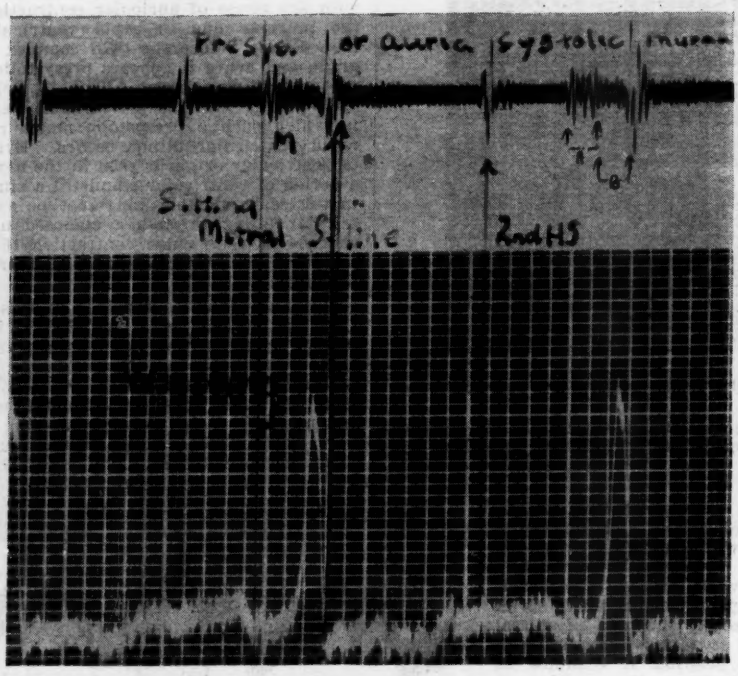

FIGURE X.
Mid-diastolic murmur and associated auricular systolic murmur in mitral stenosis and normal rhythm.

These same authors subscribe to the view that the normal third heart sound is produced by the "vibrations of ventricular walls in the final moments of rapid ventricular filling" (see Figure I).

In the present tracing the onset of the supernumerary sound is 0·12 second after the second heart sound. This makes it unlikely to be due to the opening snap of the mitral valve, as 0·11 second's interval is the upper limit for this to occur (Orias and Braun-Mendendez).

Is it then triple rhythm Type I*b* (Evans)? This appears most likely, as a triple rhythm is a common finding in mitral stenosis, especially early mitral stenosis, and the third sound is the forerunner of a mid-diastolic murmur (Levine, 1945). Bramwell (1935) agrees that in mitral stenosis accentuation of the third heart sound is the usual finding, but considers that it differs from "gallop rhythm" (left ventricular type) in its production. Bramwell maintains that both are due to rapid filling through the mitral valves and that the former is independent of auricular contraction; the latter, however, requires auricular systole for its production and is not heard in auricular fibrillation.

Mention was made above of the opening snap of the mitral valve. From studies made by Margolies and others (Margolies and Wolferth, 1933) "it is evident that in a high proportion of patients with mitral stenosis, sounds are produced at the moment in which the auricular ventricular valves open. These sounds originate in the mitral valve and are undoubtedly due to the structural changes produced by this disease. By their location in

the cardiac cycle and the acoustic characteristics which they often possess they well deserve the term suggested by Potain (1875). The opening snap of the mitral valve is an important auscultatory sign in mitral disease and may even be the only auscultatory sign by which the correct diagnosis of mitral stenosis can be established."

In Figure VI is reproduced a phonocardiogram in which Orias and Braun-Menendez demonstrate three heart sounds from a healthy pregnant woman. The phonocardiogram shows a physiological auricular sound and a physiological third heart sound. Compare this figure with Figure VII, also taken from the same work. It is apparent that it is quite impossible to distinguish this supernumerary sound with the electrocardiogram as reference alone, yet they are quite distinct, as shown by reference to the phlebogram. One is due to a normal third heart sound coinciding with the descending limb of the *v* wave and the other to the opening snap of the mitral valve coinciding with the apex of the *v* wave.

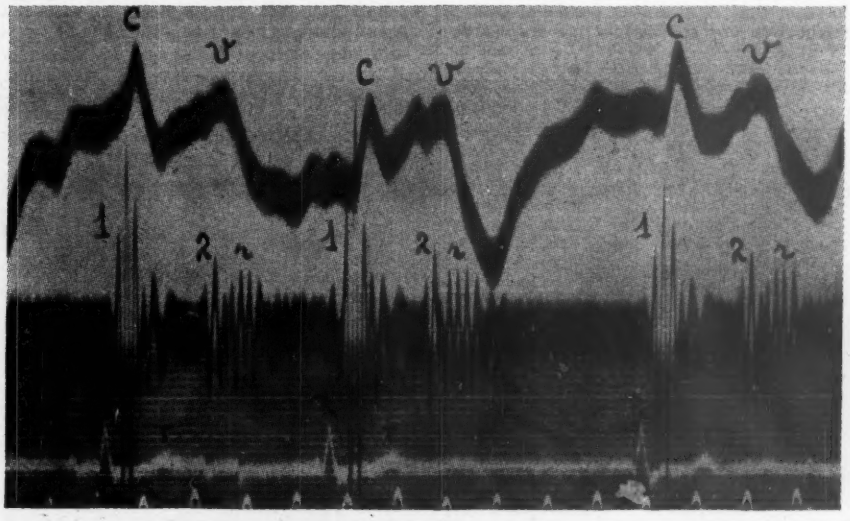

FIGURE XI.

Heart sounds in mitral stenosis, recorded at the mitral area. The diastolic rumble occupies only the rapid ventricular filling phase and illustrates the possibility of a mitral diastolic murmur occupying the early part of diastole (Battro and Braun-Menendez).

Figure VIII.

E.D., a female, aged seventy-three years, has had hypertension and congestive failure for several years. Her blood pressure is 190 millimetres of mercury (systolic) and 120 millimetres (diastolic). X-ray examination shows cardiac enlargement. An electrocardiogram reveals gross coronary insufficiency. Clinically there is a triple rhythm at the apex of presystolic type.

Comment.—The phonocardiogram reveals a triple rhythm and a short systolic murmur terminating at about mid-systole. Evans states that "when heart failure makes its appearance in hypertension, triple rhythm is commonplace, but it is usually due to the presence of the fourth heart sound [Type II*b*] and not the third" (Type I*b*). This fourth heart sound he places in auricular systole, and he states that it occurs in left ventricular failure generally before signs of right heart failure appear, namely, enlargement of the liver, ascites and œdema of the ankles. Once these signs appear, Evans maintains, the triple rhythm is generally due to the presence of the third heart sound (Type I*b*) and occasionally to a summation gallop (Types I*b* and II*b*) when tachycardia is present. After treatment and rest one type often changes to the other. Among Evans's tracings there is an example of this effect of rest and treatment. If this clinical observation is correct and the fourth heart sound in hypertension is the "cry of the heart for help" (Obrastzow, 1905), then there is clinical significance in the nature of this interpolated sound.

Is the interpolated sound in Figure VIII of the nature of a third heart sound (Type I*b* of Evans) or is it an interpolated fourth sound due to auricular systole (Type II*b* of Evans)?

By inspection it appears during the time of auricular systole, suggesting its origin as the fourth heart sound (the triple rhythm Type II*b* of Evans) and having sinister significance. But of this we cannot be sure, as our reference is the electrocardiogram and not the phlebogram, which would localize it exactly. If it was the fourth heart sound it would correspond to the ascending limb of the *a* wave, whereas if it was the third heart sound it would be related to the descending limb of the *v* wave (see Figure I), and the fact that it occurs late in diastole is no proof of its origin. "The location of the sound to the beginning, middle or end of diastole does not necessarily imply that gallop is respectively a rapid filling [Type I*b*, Evans], summation [Types I*b* and II*b*, Evans], or an auricular [Type II*b*, Evans] gallop" (Orias and Braun-Menendez).

If we are to build a superstructure of clinical value on these very points we have at least to be sure that our foundation of identification is sound. Perhaps with experience we may be able to dispense with the phlebogram when we know about its behaviour in relation to the electrocardiogram and the sound tracing, but until that time arrives a considerable amount of work will surely have to be done to elucidate such problems as we have been discussing.

It is to be noted too in the present tracing that an inconstant systolic murmur of low amplitude begins probably at the *S* line and certainly before mid-systole. This is in contrast with Evans's experience that "in each of thirteen cases with hypertension the phonocardiogram showed the murmur starting in mid-systole a little way beyond the *S* line".

Figures IX and X.

Figures IX and X illustrate two similar types of phonocardiographic tracing with sounds occurring during the presystolic phase of the heart cycle. The first example (Figure IX) is taken from Orias and Braun-Menendez and illustrates the difficulty of identifying presystole on

reference to the electrocardiogram alone. The electrocardiogram in Figure IX would identify the whole of the presystolic sound as of auricular systolic origin, as the added sounds occur within the *P-R* interval. The falsity of this assumption is demonstrated from the phlebogram, which shows that the gallop is a summation of rapid ventricular filling (downstroke of the *v* wave) and auricular systole (upstroke of the *a* wave).

Figure X is recorded from a girl, aged twenty-one years, with no history of rheumatic fever but referred because of triple rhythm at the apex and a doubtful presystolic murmur found at routine examination. X-ray films and fluoroscopy showed no enlargement of the heart. There was a history of "growing pains" as a child.

Examination of the tracing shows a "mid-diastolic" murmur with possibly another element in the presystolic period. These have been denoted by "A" and "B". It is probably fairly certain that B is due to auricular systole, but without a phlebogram one cannot identify A. Perhaps it is all part of auricular systole, but more likely it is due to a summation, and that portion or all of A is due to rapid ventricular filling.

Analysis, however, shows that to claim the constant presence of a mid-diastolic murmur as confirmation of the presence of mitral stenosis is invalid. For example, in Figure XI the diastolic rumble occurs early in diastole because the rapid ventricular filling phase occurs early and takes place very shortly after the second heart sound. It is hard to reconcile with this figure Evans's statement that a "diastolic murmur of mitral stenosis may occupy the late or mid-diastolic period, but never the early part". The position it occupies is surely the position of rapid ventricular filling in diastole.

Until identification of these presystolic components in Figure X is positive, diagnosis remains uncertain and the phonocardiogram cannot assist the clinician.

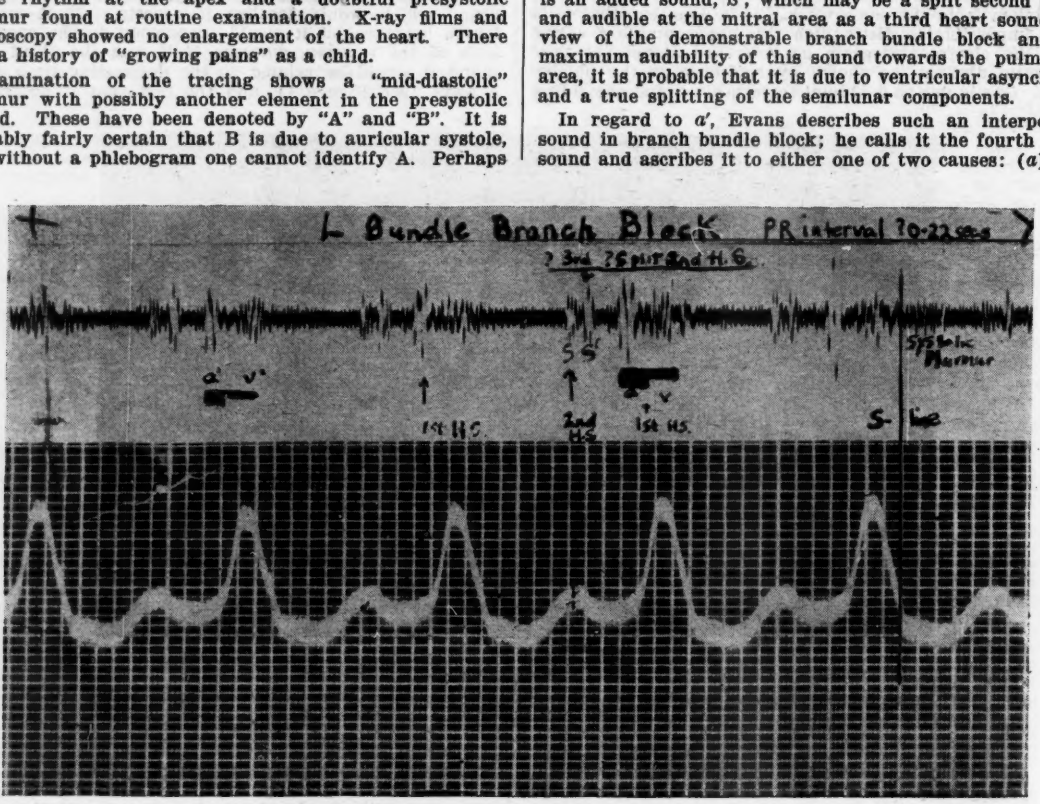

FIGURE XII. Left branch bundle block. Recorded at the mitral area.

Left Branch Bundle Block.

Figure XII.

E.T., a female, aged sixty years, suffered a coronary occlusion four years ago, following which congestive cardiac failure developed. Examination discloses the apex beat four inches from the mid-sternal line in the fifth intercostal space. The blood pressure is 160 millimetres of mercury (systolic) and 110 millimetres (diastolic). Moderate œdema of both extremities and over the sacrum is present. The pulse rate is 136 per minute. X-ray examination of heart and lungs shows transverse enlargement of the heart with pulmonary congestive changes. Auscultation of the heart reveals what may be quadruple rhythm with reduplication of the first and second heart sounds, a systolic murmur at the apex, and a possible diastolic murmur at the pulmonary area. An electrocardiogram shows left branch bundle block.

Comment.—Following the second sound (see Figure XII) is an added sound, *S'*, which may be a split second sound and audible at the mitral area as a third heart sound. In view of the demonstrable branch bundle block and the maximum audibility of this sound towards the pulmonary area, it is probable that it is due to ventricular asynchrony and a true splitting of the semilunar components.

In regard to *a'*, Evans describes such an interpolated sound in branch bundle block; he calls it the fourth heart sound and ascribes it to either one of two causes: (*a*) concomitant prolongation of the *P-R* interval (Type II*a*) or (*b*) left ventricular failure (Type II*b*). In this case, although there is early congestive failure, the *P-R* interval is just within normal limits (0·22 second) and therefore, if Evans is correct, this added sound is due to the left ventricular failure (Type II*b*) and not directly to branch bundle block.

Orias and Braun-Menendez demonstrate wide separation of the components of the first heart sound due to asymmetry of the phases (isometric and ejection) of the right and left sides of the heart.

Could *a'* therefore be due to isometric contraction of the right ventricle, which can be assumed to contract first in left branch bundle block? If this is an isometric contraction of the right ventricle, why is it so loud in view of the fact that it is not until the ejection phase that reinforcement occurs by summation with the isometric phase of the left ventricle? And again, *a'* falls even before the electrical ventricular excitation. Our conclusion, therefore, is that *a'* is pre-ventricular in origin and is not due to the separation suggested by Orias and Braun-Menendez, that it occurs during auricular systole and that it is the fourth heart sound Evans describes. This should disappear on auricular fibrillation. Unfortunately we have no tracing

to compare with this of left branch bundle block with auricular fibrillation. We should also like to see a phlebogram prepared from this patient and to determine the precise relationship of *a'* to rapid ventricular filling. If indeed *a'* coincides with rapid ventricular filling, then *a'* is not the fourth heart sound, but the third, it has nothing to do with auricular systole, and it should theoretically occur in fibrillation.

COMMENT AND CONCLUSIONS.

Phonocardiography is not a new science, but it is undergoing a rebirth. The work of Evans in England has given great impetus to the revival of this long-neglected instrument. However, much more work has yet to be done on the correlation of the clinical side with the mechanical before the phonocardiograph can be accepted as a trustworthy aid in the elucidation of cardiac problems. In this review we have attempted to show the difficulties attendant upon accepting tracings recorded under non-standard conditions with machines of unknown acoustic reliability. Little knowledge is nowhere more dangerous than in the interpretation of phonocardiograms.

Some of our results have been at variance with the findings of William Evans, possibly owing to the difference in machines and to our own inexperience, but it shows the importance of being thoroughly critical in all aspects of the work lest false conclusions be deduced from the tracings.

One point we feel should be stressed, and that is the necessity for the simultaneous recording of the phlebogram as the only accurate method of knowing what is taking place when sounds occur in the diastolic phase. The electrocardiogram as a reference is too vague to be of help.

We submit the following suggestions:

1. The technician recording phonocardiograms should be thoroughly conversant with all aspects of the necessary technique.

2. The machine should be of the stethoscopic variety, preferably with a series of frequency bands covering the useful range, and it should be reliably built.

3. Phlebograms from the jugular pulse should be the routine reference tracings.

4. The optimum chest-piece for the murmur should be used and the pressure on the chest should be standardized.

ACKNOWLEDGEMENTS.

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THE PAINFUL STIFF SHOULDER.

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THE painful shoulder is a condition commonly encountered, and presents a problem bristling with many difficulties. In order to try to do justice to the subject in this short talk, I shall confine my attention to pain in the shoulder associated with definite limitation of movement at this joint, thus excluding from the discussion brachial neuritis, arthritis and periarthritis of the cervical part of the spine, cervical disk syndrome, and pain referred from visceral disease.

The stiff, painful shoulder is of peculiar interest to me for several reasons. Firstly, it is one of the most common conditions which we encounter in the field of physical medicine. Secondly, there is a great deal of controversy, not only with regard to the pathology of the condition, but also about the correct treatment in many cases. Thirdly, it offers an outstanding example of the value of preventive medicine. The institution of efficient treatment at an early stage would, in many cases, prevent not only untold suffering, but also permanent disability.

The painful stiff shoulder may be acute or chronic. In the acute case it may be due to a variety of causes; but whatever the cause, there is one cardinal principle in treatment and that is the commencement of active movement as early as possible, in order to prevent the formation of adhesions and the development of a "frozen" shoulder—a nightmare often to patient and doctor alike.

Arthritis.

Arthritis of the shoulder is relatively uncommon. It is characterized by acute pain and tenderness over the joint, particularly anteriorly, with painful limitation of movement in all directions. The pain is usually severe at night. Radiological examination may be helpful. If the condition is tuberculous or pyogenic, surgical measures are required. Rheumatoid arthritis is rarely seen in the shoulder, until other joints have become involved, and the treatment is directed accordingly—removal of septic foci, which are diligently sought, whether in teeth, tonsils, antra, genito-urinary tract, bowel *et cetera*. In the absence of apparent foci, chrysotherapy is usually employed, preferably with small doses of "Myocrisin", together with general medical treatment such as an adequate diet rich in vitamins and attention to anæmia if present, *et cetera*. Physical measures are employed for traumatic arthritis.

Traumatic arthritis is also relatively uncommon. Treatment consists of rest and the application of heat—preferably penetrating heat such as short-wave therapy or diathermy. When the acuteness subsides, assisted active movements are commenced to restore mobility. Electrical stimulation employed carefully at an early stage will prevent muscle wasting, and assist restoration of power.

Fractures.

Fractures in the region of the shoulder joint are characterized by local signs and will not be missed if routine radiological examination is carried out in all cases of acute painful shoulder of traumatic origin. After reduction and immobilization, as soon as clinical union is evident, mobilization should be commenced. Short-wave therapy is the most effective form of heat, and combined with massage and assisted active movements, administered at least three times a week, will gradually restore function.

In the case of impacted fracture of the neck of the humerus, these physical measures may be started carefully in the first week. Time is the essence of the contract, and the longer the delay in starting mobilization, the more difficult becomes the problem of rehabilitation. Electrical stimulation may often be started before splints are removed, if there is any sign of muscle wasting. If the injury is complicated by a nerve lesion such as deltoid palsy, electrical stimulation, together with reeducation exercises, will hasten the recovery of full power. In severe deltoid palsy, an abduction splint will be necessary to prevent overstretching of the muscle.

Dislocations.

Dislocations at the acromio-clavicular joint or the glenoid cavity are characterized by local tenderness, pain and deformity; and the diagnosis is confirmed by radiological examination. After reduction and immobilization for some weeks, early mobilization is commenced as with fractures, care being taken to try to prevent redislocation. In the early stages of mobilization after dislocation at the glenoid cavity, it is wise to avoid external rotation, which is particularly prone to cause redislocation.

Tendinitis.

There is one particular type of lesion which is of considerable importance, and this is tendinitis. Codman (1934) in America pioneered the study of this condition and showed that the condition resulted from the peculiar anatomy of the shoulder joint. The subscapularis in front, the supraspinatus above and the infraspinatus and *teres minor* behind, have a common tendon of insertion with which is closely associated the long head of biceps. The supraspinatus muscle and tendon are really the key to the pathology of the region. They are separated from the acromion above by the subacromial bursa, which serves to facilitate the movement of the tendon beneath the acromion process. This tendinous region is subject to constant movement and strain, and a comparatively mild trauma will be perpetuated by the constantly recurring motion and friction. This trauma may be quite mild—exposure to cold or damp may be sufficient.

There are several varieties of tendinitis, and I shall deal with the more acute types first.

Rupture of the Supraspinatus.

Rupture, partial or complete, of the supraspinatus may be spontaneous, but usually follows a strain or injury. Even in these traumatic cases, however, some prior, degeneration of the tendon is apparently a factor, since the condition rarely occurs in patients aged under forty years, and is most common above this age. The patient, usually a labourer, complains that while lifting, he felt or heard a snap in his shoulder, accompanied by a sudden sharp pain in the tip of his shoulder. The initial trauma may vary; it is usually sudden in nature, but may not necessarily have been severe. He notices inability, partial or complete, to lift his arm up. The pain gradually extends down to the region of the deltoid insertion.

If the rupture is complete, there is complete inability to raise the arm, even if this is tested with the patient supine to eliminate gravity. Local tenderness is present over the tendon. In complete lesions, surgical intervention is called for, followed by immobilization with the arm in abduction until healing has occurred.

Usually the rupture is partial, and if it is of minor extent, diagnosis may be difficult. In such cases Dawborn's sign is of value. This is characterized by a "catch" pain, experienced on raising or lowering the arm, and felt only while the arm is moving in the arc from 60° to 120°. In partial cases of this nature, conservative treatment is usually sufficient. An injection of "Novocain" into the local tender area will often completely abolish the pain, and penetrating heat in the form of diathermy or short-wave therapy is commenced at once. Massage is not used as a rule, and in mild cases a few active movements are started with the patient in the supine position, to remove the load of gravity. These movements are then gradually increased. In more severe cases it is necessary to use an abduction splint at first, but it is wise to avoid this if possible, as patients do not take kindly to it.

Rupture of the Long Head of the Biceps.

In rupture, partial or complete, of the long head of biceps, again degenerative changes usually precede the injury and one day while wielding a hammer, the patient suddenly feels a sharp snap, with pain in his arm, and a sudden loss of power. Local tenderness is present, with weakness of elbow flexion, and a lump can be seen and felt in the arm. If the rupture is complete, surgical repair is indicated, followed by immobilization for six to eight weeks, and then graduated mobilization is commenced. If the rupture is incomplete, the signs are less clear. Surgery can here be avoided, but immobilization is desirable for several weeks before movement is commenced.

Tendinitis Alone.

The onset may be acute or chronic, consisting of pain at the tip of the shoulder, associated with local tenderness over the supraspinatus tendon or the long head of biceps. The pain may radiate to the deltoid insertion, and sometimes palpable crepitus is present. There may be a history of trauma; if so it is usually mild in degree, and it may consist simply of exposure to cold; but often there is no history of trauma. Focal infection may play a part, especially in recurrent tendinitis, and I have seen such a condition cleared up by the removal of septic foci. Watson-Jones and others regard most of these cases as of subacromial bursitis; on the other hand, Codman and his followers regard them as of primary tendinitis, any bursitis if present being thought to be secondary; I myself incline to the latter view. This condition is often associated with coronary disease and Codman's theory offers an explanation by assuming that such a patient tends to hold his arm in adduction during attacks of angina, and that the extra strain involved sets up tendinitis.

Treatment in such cases consists in the application of penetrating heat, and the avoidance of strain. Sufficient active movements are given to prevent the formation of adhesions, and recovery usually occurs in a few weeks. If pain is very severe and not responding to treatment, the use of deep X-ray therapy is sometimes of value.

Tendinitis, Associated with Calcification.

The picture in tendinitis associated with calcification is almost the same as that of tendinitis alone except that the degeneration has proceeded further in the tendon, with the deposition of calcium salts, usually as calcium phosphate and calcium carbonate, a soft cheesy mass being formed in the supraspinatus tendon or the subacromial bursa. The presence of such calcification should always be suspected if there is a history of recurrent attacks and it can be confirmed radiologically. Short-wave diathermy, three times a week, together with a few active movements to prevent the formation of adhesions, usually removes the pain after about six or eight weeks and further X-ray examination will often reveal complete disappearance of the calcification.

In tendinitis with or without calcification, Dawborn's sign is often helpful in the diagnosis.

Fulminating Subacromial Bursitis.

In my experience fulminating subacromial bursitis is uncommon; but its recognition is important, since the treatment is different. There is an acute onset, with or without mild trauma, of sudden severe pain at the tip of the shoulder. Localized tenderness and often visible swelling are present just below the acromion process, and movement of the shoulder is acutely painful. Heat usually aggravates this condition. I have found the most effective measure is the intermittent application of an ice bag for twenty-four hours. This usually produces dramatic relief, apparently by relieving tension inside the bursa. Complete rest and sedatives are used for a few days, and then a cautious commencement is made with the application of mild heat and a few active movements, this regime being carefully increased in degrees until a full recovery is made

in a few weeks. Anodal galvanism is often a valuable alternative method for the first few days. Deep X-ray therapy is sometimes used, but I consider it inferior to the other two methods.

Fibrositis.

A common cause of painful stiff shoulder is fibrositis, which may be traumatic or rheumatic. In such cases local pain and tenderness are present over the affected muscle, and movements involving this muscle will be limited whereas movements in other directions are free. Injection of 0·5% "Novocain" solution into such a localized area will give considerable relief at once; but this is often only temporary. However, if this is followed up by the application of heat, massage and active movements, a cure rapidly results. Massage should be gentle at first, and as the acuteness subsides a more kneading type of massage is used. Without this deep massage the condition may be slow in completely resolving; on the other hand, if too much pressure is used, and at too frequent intervals, an exacerbation results. In my opinion considerable judgement is necessary with regard to the frequency and depth of massage, and lack of appreciation of this fact is responsible for many failures.

In many cases the pain is referred from a fibrositic nodule some distance away from the site of pain. Shoulder pain is often referred from nodules in the trapezius or infrascapular region, for example. This has been demonstrated by Kelly (1942). A careful search should be made for such nodules. The diagnosis is confirmed when injection of such nodules abolishes the pain at once.

In rheumatic fibrositis, apart from local measures, a search should be made for septic foci, and these should be eliminated. Attention must also be directed to other medical measures, such as diet, habits of living and the treatment of psychiatric disturbances.

Chronic Shoulder Stiffness.

These various types of acute painful shoulder if neglected may merge into the chronic condition.

In the vast majority of cases the chronic condition falls into the group known as periarthritis of the shoulder, or Duplay's syndrome, which presents a characteristic picture. The history given is that of a gradual onset extending over weeks or months of pain in the shoulder, often most pronounced over the region of the deltoid insertion. The pain often radiates down the arm to the hand, and is often worse at night. It is rarely accompanied by paræsthesia, unlike other types of brachial neuralgia. Coincidently with the development of pain, the patient usually notices a progressive diminution of movement at the shoulder, especially in abduction. Nevertheless it is surprising how a pronounced limitation of movement may pass unnoticed by the patient. The signs are characteristic. There is limitation of movement in abduction and rotation to both active and passive movements and extremes of movement cause pain. These signs may be so pronounced that the shoulder joint is completely fixed—the "frozen" shoulder—but the X-ray picture reveals no abnormality. This condition is one of inflammation of the periarticular tissues, and may have a variable pathogenesis. In many cases the initiating cause is trauma—perhaps a mild strain or even exposure to cold. Because of pain the patient limits movement and adhesions soon form and a vicious circle is set up, with progressive limitation and pain. In some cases the initiating trauma has been such as to necessitate immobilization for some weeks—for example, traumatic arthritis, fracture and dislocation, and in such cases some resultant stiffness of the shoulder is unavoidable; but in many cases it is preventible by early mobilization. "A movement a day keeps adhesions away." Lack of this precaution produces the "hemiplegic shoulder" and undue use of the sling in upper limb injuries is another common cause. We still see cases of Colles's fracture in which a "frozen" shoulder has developed from lack of appreciation of this truism.

In other cases the initiating cause is a rheumatic affection in the region of the shoulder—perhaps simple fibrositis which the patient feels is insufficiently serious to justify medical attention, and he may not seek advice until considerable disability has occurred.

In cases in which there is no history of an injury or of a rheumatic attack, the condition has probably started as tendinitis in which the trauma has been mild as in the cases that are found in association with coronary disease. Codman states that in these cases the frequent adduction posture of the arm in anginal attacks imposes undue strain on the tendons. In cases in which there is a definite history of injury, the prognosis is much better usually than in other types.

The most important therapeutic measure is obviously prevention—prompt and efficient treatment of all shoulder injuries, even if they are only mild—and early mobilization will prevent the development of this painful disability.

However, the treatment of the established condition is more difficult; it requires patience, perseverance and experience, together with the full cooperation of the patient. Moreover, there is considerable controversy about the best method of attack and there are two main schools of thought.

The radical school, exemplified by Douthwaite (1938), believes that in all established cases the adhesions should first be broken down by an initial manipulation under general anæsthesia. This formidable procedure is then followed up by physical measures of heat, massage and exercises. Douthwaite states that conservative methods alone will fail, and many surgeons still believe in an initial manipulation under general anæsthesia. With this view I am in complete disagreement, and this for two reasons. Firstly, in my experience the majority of these patients do well without recourse to manipulation under general anæsthesia. Secondly, such manipulation is followed by a severe reaction causing much pain, not only immediately afterwards, but during the follow-up period of massage and exercises. I do not, of course, dispute that in some cases such manipulation is necessary; but I do condemn its indiscriminate use in all cases. I fully subscribe to the views expressed by Watson-Jones (1938*a*): "Manipulation under anæsthesia should not be used except as the last line of treatment." Further, I believe that when it is performed, too much is often attempted at the one time. It is often wiser to do less, and repeat the manipulation as necessary. Watson-Jones again expresses this view in the following words: "Not more than one group of adhesions must be broken at a time. If too much is done the reaction is so severe that the movement can not be retained, and the joint may become stiffer."

Codman (1934) and McKenna (1938) prefer to put the patient in bed for a few days, using strapping extension and traction to restore movement gradually, combined with radiant heat and sedatives.

The procedure I use at present is to treat the patient three times a week with short-wave therapy, followed by massage and assisted active exercises. The short-wave therapy not only helps relieve pain, it also facilitates massage and movement. Massage should be directed at the muscles and not forcibly over the joint, as this will only increase pain. Analgesic drugs are necessary to relieve pain, with the use of heat at home. Active exercise is the most important factor in success, and right from the outset it must be impressed on the patient that he should persist in his active exercises despite pain, and that his pain will not diminish until his movement increases. On his perseverance in persisting with active movements regularly despite pain depends his result.

The range of movement is recorded each week, and usually in six or eight weeks the range is fairly good, and pain is much less. In some cases treatment is necessary for a further month or two before a satisfactory result is obtained.

Manipulation under anæsthesia is considered, (i) if after one month or six weeks of treatment there is no pronounced increase in the range of movement, and (ii) if movement ceases to improve, and remains at a standstill. The procedure is best avoided if concurrent osteoarthritic changes are present, or if the patient is unable to lie on the shoulder at night. If there is an active focus of infection—

in teeth or elsewhere—it should be dealt with at the start of treatment, as it may aggravate the condition.

It is wiser to carry out two or three small manipulations rather than a single manipulation so extensive that the reaction is so severe that movement cannot be maintained.

In all cases it is a *sine qua non* that the manipulation be immediately followed up with daily application of heat, massage and active movements to retain the increase in range, and sedative drugs are often necessary to control pain.

Deep X-ray therapy is sometimes of value in cases in which pain is is severe and persistent. When pain has eased after a week or two, a gradual start may be made with the usual measures.

Occasionally, particularly in aged patients, recovery may not be complete, there being some residual pain and limitation of movement; but this is uncommon in uncomplicated cases. A less common cause of chronic stiff painful shoulder is osteoarthritis. This is seen most often at the acromio-clavicular joint. It presents a similar picture to periarthritis, but sometimes creaking can be detected over the joint on movement. This is suggestive but not conclusive, as a similar creaking can occur from thickening in the subacromial bursa which is sometimes found in periarthritis. The X-ray picture is of course conclusive. Similar treatment is given as in periarthritis, but it is usually necessary for several months, and although it is often successful, relief is sometimes only partial.

Osteoarthritis at the glenoid cavity is confirmed by radiography, but is fortunately very rare, as it is refractory to treatment in many cases. In any case, treatment is necessary for many months.

In cases of ostearthritis injury may suddenly light up the condition, simulating an acute onset.

Calcification in the supraspinatus tendon or in the subacromial bursa may cause chronic pain, or reccurrent attacks. The pain is usually over the tip of the shoulder, and Dawborn's sign is sometimes present, assisting in the diagnosis; however, this is made only on radiological evidence.

Routine X-ray examination of all stiff, painful shoulders will enable the diagnosis of calcification to be made with accuracy and reliability. The use of short-wave therapy or diathermy, together with active exercises, three times a week, will usually effect a cure in about two months. At this time a check X-ray film will often reveal that the calcium deposited has been completely absorbed. Deep X-ray therapy is sometimes used as an alternative method of treatment. Surgical measures, such as irrigation by means of large bore needles, needling alone, or even actual excision, may be employed, but are usually unnecessary except in extensive cases.

Conclusion.

In conclusion, let me point out that the most common and troublesome type of stiff, painful shoulder is that due to periarthritis. The most valuable form of treatment is prevention and the early and efficient treatment of all painful shoulders—even if only minor in degree—will usually prevent the development of this painful and crippling disability. The early institution of active movements is most important, and this is facilitated by pain-relieving measures such as the application of penetrating heat, "Novocain" injections into localized areas of tenderness, sedative massage and sedative drugs. "A movement a day keeps adhesions away." As Watson-Jones puts it (1938*b*): "If immobilization is not essential, then mobilization is imperative."

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THE MACROSCOPIC DIAGNOSIS OF MALIGNANCY IN OVARIAN TUMOURS.

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WHILE the microscopic analysis of ovarian tumours is one of the most fascinating objects to the pathologist, such study is of no great help to the gynæcological surgeon, whose main concern it is to decide at the operating table whether a tumour which he has just exposed is benign or malignant. On this decision depends the further course of the operation—whether a simple removal of the tumour will be sufficient, whether the ovary may have to be removed as well, and whether a hysterectomy will have to be performed. Therefore, the macroscopic criteria of malignancy in ovarian tumours will be the subject of the present discussion.

A word may be said in passing about the advisability of having immediate frozen sections of all ovarian tumours examined during operation. While there is no doubt that this can be successfully done under ideal circumstances, there is also no doubt that from numerous points of view such a procedure is impracticable as well as inadvisable. First of all, there are not many pathologists who have such an experience in the diagnosis of ovarian tumours that they can make in practically all cases a "spot diagnosis" with a sufficient degree of certainty. Even if such pathologists were generally available, it would be a question whether their time would not be too valuable to be taken up with unavoidable waiting in the operating theatre, and whether their work in general would not suffer from too frequent interruptions. However, one much more serious consideration concerns our teaching hospitals; it is, I think, wrong in principle to make surgeons used to facilities which will be available to them only under exceptional circumstances. There can be no doubt that clinical acumen would suffer greatly if, in the teaching hospitals, the responsibility for the decision whether a tumour was likely to be malignant or not was taken away from the surgeon in the operating theatre and placed on the shoulders of the pathologist. On the other hand, most ovarian tumours display sufficient criteria for this vital decision, as will be seen in the following paragraphs.

For a proper examination of a tumour, it will be necessary, after inspection of the outside, to give careful attention to the cut surface. This should be prepared, not by an odd incision into the tumour with a scalpel, but, as the preservation of the specimen for museum or teaching purposes should be kept in mind, by a clean bisecting cut with a long knife (amputation knife). This can often be done by an assistant who does not take part in the operation. It may, however, as will be seen later, be necessary on occasions for the surgeon to examine himself, by touch, certain properties of the tumour, even if this entails a change of gloves and other precautions against implanting tumour fragments into the field of operation.

Of the points that assist in the diagnosis of malignancy or otherwise, the question of the size of the tumour may be discussed first. This is, in general, not a helpful point; but one can say that an extraordinarily large tumour is most likely to be benign (a malignant tumour kills the patient before it has time to attain really large size). A suspicion of malignancy is always raised if the history contains a note of a recent sudden increase of the size of the tumour.

Next, attention will be paid to whether the tumour is cystic or solid. Of the cystic tumours some will be cystic throughout, others will be filled to varying degrees by papillary formations, still others will contain more or less solid areas within the cystic formations.

A tumour that is cystic throughout, whether unilocular or multilocular, is likely to be benign. Often the cystic contents lead to accurate diagnosis of the tumour—as, for example, in the case of a pseudomucinous cystadenoma.

In those cases in which papillary formations are present, much depends on the characteristics of these papillæ. In some cases, one finds fairly flat, rounded, hard protuberances covering certain portions of the walls; such tumours are benign. If there is luxuriant papillary growth filling considerable portions of the cystic tumour, one can say generally that the finer, more delicate and more dendritic the papillæ are, the more likely it is that the tumour is benign. If the papillæ become more succulent, softish, larger and friable, then the suspicion of malignancy arises. It is of no significance if some of the papillary growth appears on the outside of the cyst. This may or may not indicate malignancy; the decision depends on the characteristics of the papillary growth just enumerated.

Cystic tumours which contain more or less solid portions are likely to be malignant. There are perhaps two exceptions to this general rule, and both concern tumours which are fundamentally pseudomucinous cystadenomata. A few of them may be of the extreme microcystic type—that is, they may be composed of innumerable very small cysts which are filled with very thick pseudomucin. In this way a solid appearance of portions of such a tumour may be brought about; but a hearty squeeze will reveal the true state of affairs by the appearance, on the cut surface, of comedo-like extrusions of the cystic contents. In other rare cases, a whitish, hard nodule may be found in a pseudomucinous cystadenoma and will prove on microscopic examination to be a benign Brenner tumour. However, except for such rare cases, the finding of solid tumour tissue within cystic neoplasms is a definite indication of malignancy.

If an ovarian tumour proves on bisection to be solid and not cystic, palpation will be necessary to decide whether it is hard or soft. A hard tumour is probably benign; if it has a whitish cut surface with rather definite whorling, it is sure to be a benign fibroma or fibromyoma, if it is yellowish in colour, an equally benign theca-cell tumour comes under consideration.

Of the soft solid tumours there is one that can be accurately diagnosed by inspection and touch with a high degree of certainty, and that is the dysgerminoma. Instead of a lengthy description, it is probably best to say that it looks and feels like brain tissue. However, it is unfortunate that no definite statement with regard to its malignancy can be made. Still it is certain that such a tumour is in general much less malignant than its counterpart in the testis, there often called seminoma. It is therefore probably justified, especially if (as so often happens) the tumour occurs in young women, to be content with the simple removal of the tumour, if it is well encapsulated and not adherent to surrounding structures.

Almost all the other soft solid tumours of the ovary will be malignant, especially if the tumour tissue is not only soft but friable. On some occasions, an apparent softness may be due to a high degree of œdema of an originally hard tumour. However, such a benign tumour will give a feeling of elasticity to palpation, while the exploring finger will break through the surface of a friable malignant tumour.

Another point worthy of consideration is whether only one or both ovaries are involved in tumour formation. A unilateral tumour is not necessarily benign, but a bilateral tumour is always suspect of malignancy.

Extensive adhesions to surrounding structures will raise the suspicion that one is not dealing with a benign tumour; secondary involvement of such structures proves malignancy. At all operations for ovarian tumours a careful inspection of the pelvis and of as much of the abdominal cavity as possible should be carried out. If secondary deposits are found, there can be no doubt about the diagnosis.

So far, discussion has centred around the straightforward question whether a certain tumour is benign or malignant. While this decision is of the utmost importance, it would be of considerable advantage if one could assess sometimes the degree of malignancy of certain malignant tumours. This is especially so if the woman is young and desirous of having a family. The macroscopic examination of a tumour is usually not sufficient; for this purpose, however, there are sometimes additional factors either in the structure of the tumour or in the history of the patient that will be of help. The case of the dysgerminoma has already been discussed. Furthermore, it should be remembered that those solid ovarian tumours that have indicated their presence by hormonal disturbances are usually only of limited malignancy, and in such patients a non-radical operation may occasionally be justified by the circumstances. Malignant cystic tumours are usually thoroughly malignant and require complete operation. The question of the malignancy of a more or less solid teratoma is of interest. While a solid teratoma of the testis is always malignant, this is not necessarily so in the ovary. There are ovarian teratomata in which all the various tissue elements are fully differentiated. Such tumours will not recur after simple removal. There is, however, no way of recognizing this at operation, and a complete operation is therefore preferable.

A special word may be said about the significance of ascites in conjunction with ovarian tumours. It is true that ascites often occurs in the presence of malignant ovarian neoplasms, but the former attitude that ascites invariably indicates such an advanced stage of a malignant condition that it has become inoperable, is no longer justified. First of all, it has been shown that ascites can occur with completely benign ovarian tumours of the fibroma type (Meigs's syndrome). However, it is still more important that ascites occurs with certain papilliferous cystadenomata of the ovary, especially those that show papillary growth on the outside of the cyst. In such cases, it may happen that fragments from the papillary formations are shed and gain a foothold on the omentum or other peritoneal surfaces. Thus, in some such cases the finding of ascites and of secondary deposits all over the peritoneum has led to the closure of the abdomen without any attempt to remove the primary tumour. This is fundamentally wrong; it may not always be possible to remove the primary growth, but an attempt should be made, because it has been shown that in cases of benign primary tumour this secondary sowing can also be benign in character; that is, the secondary deposits will remain superficial, will not grow into the underlying structures, will not lead to the general consequences of malignant growth, and will even disappear after removal of the primary tumour. It must be understood that such cases are very rare indeed—the vast majority with similar findings will prove to be malignant; the point has been stressed only for the reason that in all cases a removal of the primary tumour should be attempted, since in the past patients have died from peritonitis that developed after repeated tapping for ascites due to a benign growth.

Lastly, a point may be discussed which is not directly concerned with the question of malignancy of an ovarian tumour, but which often gives rise to confusion. Most text-books stress that the hormonally active tumours of the granulosa-cell and theca-cell group are characterized by causing irregular uterine hæmorrhage of the *metropathia hæmorrhagica* type. However, a number of patients who are found to have a growth of this kind report sometimes long periods of amenorrhœa. It should be realized that at certain levels of hormone production by such tumours an equilibrium between pituitary and ovaries is reached which abolishes the fluctuations of the œstrogen levels which in turn are responsible for the episodes of bleeding. A history of amenorrhœa, therefore, does not rule out the presence of a granulosa-cell or theca-cell tumour of the ovary.

If the criteria which have just been discussed are given careful consideration, in most cases it will be possible to arrive at the decision whether an ovarian tumour is benign or malignant. In the few in which no definite conclusion can be reached, it will be generally advisable to carry out the most radical operative procedures compatible with the special circumstances. In every case, even if the growth appears to be hopelessly malignant, an attempt at its removal should be made, and in some cases the effort will be rewarded beyond expectation.

A STUDY OF HEALED FRACTURES IN THE AUSTRALIAN ABORIGINAL.

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DURING 1947 a survey was made of the aboriginal skeletal material housed in the South Australian Museum. This report deals with the incidence of healed fractures of aboriginal bones with comments on certain items of interest which emerged from the study.

It would appear that the aboriginal, even if he is able to recognize gross fractures, has no knowledge of the value of splinting, and J. R. B. Love (1939) states definitely that the aboriginal is totally ignorant of bone setting. Apparently, though, the aboriginal realizes that bones can be fractured, as is evidenced by the practice of forcibly fracturing the humeri of his womenfolk as a punishment. It is therefore interesting to find that examination of many of these fractures shows anatomical restoration that would be considered good even by modern orthopædic standards.

It must be pointed out that no conclusions can be drawn from this study as to the total incidence of fractures among aborigines. Presumably, many of the bone fragments in this collection represent ante-mortem fractures in cases in which the subject died as a result of the fracture or associated injuries before healing had time to be manifest. On the other hand, many of the fractures must have occurred *post mortem*—for example, from weathering, or from the point of a pick during recovery of the skeletons. The technical difficulties of separating the two classes of fractures are such that this study is restricted to a consideration of bones showing healed fractures only.

In most cases the presence of a healed fracture was made obvious by callus formation, with or without deformity. In doubtful cases the bone was radiologically examined, and it was then possible to determine the existence of a fracture from the radiological picture. Occasionally a localized patch of tertiary yaws gave appearances similar to subperiosteal fracture; but in such cases the X-ray appearance of circumscribed areas of rarefaction together with sclerosis of the cortex enabled the differentiation to be made.

The material is divisible into two large groups. On the one hand there is a collection of individual bones, numbering over 8000, while on the other hand there are 272 more or less complete aboriginal skeletons (excluding skulls).

In the first group 49 healed fractures were discovered. The distribution of bones in this group, together with the incidence of fractures of each type of bone, is shown in Table I. It will be noted from Table I that the healed fractures were confined to the long bones of the limbs, the skull and axial skeleton being free from evidence of healed fractures.

The examination of the 272 partial or complete skeletons, representing a total of approximately 30,000 bones, disclosed 45 fractures distributed as shown in Table II. Once again, it will be noted that the healed fractures are present in the long bones of the limbs only, with the exception of a solitary fractured rib.

Discussion.

The following points of interest emerged from the examination of the various groups of fractures. Uniformly obvious was the high degree of anatomical restoration. In the majority of cases the final result was good, and very few cases of gross distortion were seen. It will be noted that in the upper limb the incidence of fracture increases generally in a distal direction, while in the lower limb the incidence decreases distally.

Humerus.

Of the nine fractures of the humerus, four were right-sided and five were left-sided. Six fractures occurred in the lower third of the bone, two in the mid-shaft and one in the upper third. Alignment in all cases was good.

Radius.

Of nine fractures of the radius, two were right-sided and seven left-sided. The lower third of the bone was fractured six times, the mid-shaft twice, and the upper end once. This series included a case of bilateral healed Colles's fractures, in which some backward tilting of the radial articular surfaces, and backward displacement of the distal radial fragments, were seen together with considerable radial bowing of the lower ends of both ulnæ.

TABLE I.
Incidence of Healed Fractures Among 8368 Aboriginal Bones.

Bones.	Number Examined.	Number of Healed Fractures.
Upper limb:		
Clavicle	203	1
Scapula	216	—
Humerus	442	4
Radius	236	1
Ulna	294	32
Bones of hand	980	—
Lower limb:		
Innominate	240	—
Femur	515	7
Tibia	504	3
Fibula	254	1
Patella	63	—
Bones of foot	1210	—
Trunk:		
Vertebræ	1430	—
Sacrum	83	—
Sternum	39	—
Ribs	1024	—
Skull:		
Frontal	34	—
Temporal	38	—
Maxilla	152	—
Parietal	37	—
Occipital	294	—
Total	8368	49 (0·58%)

Ulna.

The number of fractured ulnæ (56) exceeded the total of all other fractures together (38). The reason for this becomes apparent on consideration of the actual site and nature of the fracture in relation to the probable trauma which produced it. Of the 56 fractures, 21 were right-sided and 35 were left-sided. Forty-two were two to three inches above the lower end, ten were in the mid-shaft and only three were in the upper end. There was one case of multiple fracture, to be mentioned later. X-ray films of fractures in the lower third revealed that in the majority of cases the fracture was incomplete, being restricted to the medial side of the shaft. There were, however, several good examples of complete fracture in this region. With one exception all had healed in good alignment, probably because of the splinting action of the corresponding radius. In the exceptional case there was gross forward bowing of the lower third of the ulna. Unfortunately, the companion radius of this bone was absent, so it was impossible to know whether it had been fractured also.

The appearance of the lower shaft fractures together with the left-sided predominance suggests that this type of injury may have been caused by blows transmitted to the ulnar side of the forearm by the edge of a shield, or may have occurred simply because the ulnar side of the forearm is commonly employed to ward off blows. Further evidence to support this hypothesis is afforded by charting the sites from which these bones were recovered. If it is assumed that these aborigines were buried within their tribal areas, it was found that with the exception of seven bones whose recovery site could not be determined, all the low-level ulnar fractures came from areas in which hand-to-hand fighting is customary. The location of the other bones showed considerably wider dispersal, some coming from spear-fighting areas, others from shield-fighting areas.

Femur.

Of eleven fractures of the femur, six were right-sided and five were left-sided. Six occurred in the mid-shaft, three in the upper third and two in the lower third. No transcervical or intertrochanteric fractures were seen. The anatomical result of these fractures was worse than in other bones, as would be expected from the powerful muscles attached to the femur. Angulation deformity was not pronounced, but in all specimens except one case of incomplete fracture, moderate to gross shortening was seen.

TABLE II.

Incidence of Healed Fractures Among 272 More or Less Complete Aboriginal Skeletons.

Bone.	Number Examined.	Number of Healed Fractures.
Clavicle	478	1
Humerus	422	5
Radius	496	8
Ulna	460	24
Femur	397	4
Tibia	384	2
Rib	3690	1 (Right second rib)
Total	—	45

Other Bones.

Examination of the remaining bones showing healed fractures—namely, the tibia, fibula, clavicle and ribs—revealed no particular points of interest. However, the low incidence of healed rib fracture suggests that in the majority of cases associated injury to thoracic viscera proved fatal.

Sex Incidence.

In the case of fractures occurring in the 272 skeletons, it was possible to differentiate the fractures according to sex as shown in Table III.

TABLE III.

Sex Distribution of 30 Healed Fractures of Aboriginal Bones.

Bone.	Number of Fractures.	
	Male Subjects.	Female Subjects.
Humerus	2	3
Radius	1	1
Ulna	11	8
Femur	3	—
Tibia	1	—
Totals	18	12

It was possible to determine the sex of 179 of the skeletons, the male:female sex ratio being 106:73. If it is assumed that the remaining skeletons were distributed in approximately the same sex ratio, it is found that the difference between the number of fractures in skeletons of each sex is not statistically significant; hence no conclusions can be drawn as to the relative incidence of healed fractures among male and female aborigines.

Multiple Fractures.

There were nine instances of fractures of different bones of the same skeleton with recovery. These are set out in Table IV.

In addition there was one instance of an ulna showing three fractures with healing in almost perfect alignment. Two were complete transverse fractures—one was in the mid-shaft and one was in the upper third—while the other was an incomplete fracture of the lower third, of the type which, as has been suggested, results from hand-to-hand fighting.

In none of these cases of multiple fracture was there any evidence that they were the result of bone metastases or primary bone tumour. There was, of course, no evidence either that these fractures had been sustained simultaneously as the result of a single trauma.

TABLE IV.

Bones Involved in Healed Multiple Fractures of Aborigines.

Bones Involved.	Number of Cases.
Both femora and right ulna	1
Both humeri and left clavicle	1
Both radii, right ulna and right femur	1
Both radii and both ulnæ	1
Left radius and left ulna	2
Right femur and right humerus	1
Both ulnæ	1
Left radius, right ulna and left humerus	1

Ununited Fractures.

There were two cases of ununited fracture, both occurring in ulnæ. In each case the fracture was in the mid-shaft and a false joint had been produced, the upper fragment forming the ball and the lower fragment the socket.

Summary.

Ninety-four healed fractures of aboriginal bones were examined. Apart from one fractured rib, all the fractures were confined to the long bones of the limbs. Fifty-six of the fractures occurred in the ulna, and of these 42 were in the lower third of the bone. This probably reflects the greater vulnerability of the ulna—especially the left—in hand-to-hand fighting. The majority of the fractures healed in reasonably good alignment.

Conclusion.

In a primitive society even severe bony injuries are not necessarily fatal, and without any special treatment it is possible for fractures of the long bones to heal in comparatively good alignment. The good results observed in this series represent survivals, of course, and do not preclude the possibility that a great many more fractures achieved a result bad enough to cause death before healing could ensue.

Acknowledgements.

I am indebted to Professor A. A. Abbie for suggesting the subject and for his assistance, and to the Director of the South Australian Museum for making the material so readily available.

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Reports of Cases.

TWO CASES OF FOREIGN BODY IN THE LEFT MAIN BRONCHUS IN CHILDREN.

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Sydney.

Case I.

A SMALL GIRL, aged four years, was admitted to hospital on November 18, 1948, having had a distressing cough and considerable dyspnœa for forty-eight hours. On physical examination of the patient, increased vocal fremitus and a dull percussion note were observed at the base of the right lung and bronchial breathing was heard at the base of the left lung. The temperature was normal on her admission to hospital, but rose to 100·4°F. at the end of the second

day. A provisional diagnosis of left-sided pneumonia was made and a course of sulphadiazine commenced. Neither this nor a subsequent course of penicillin produced any general improvement or prevented repeated rises of temperature occurring every few days.

X-ray examination of the chest shortly after the patient's admission to hospital (Figure I) showed apparent patchy atelectasis of the right lung, which was thought to be due either to widespread bronchopneumonia or to a foreign body impeding air entry in the right main bronchus. Examination of the chest under the fluoroscopic screen, however, showed the true nature of affairs, that is, a valvular block of the left main bronchus. The first film (Figure I) had been taken on expiration, but a fixed distension of the left lung had prevented its deflation. A to-and-fro movement of the lower part of the mediastinum was evident under the screen, movement being towards the right on expiration and back to the normal mid-line position on full inspiration (Figure II). The left dome of the diaphragm had a greatly diminished range of movement, being practically fixed at its inspiratory limit.

No direct shadow of any foreign body was seen, but the block was assumed to be due to a non-opaque foreign body in the left main bronchus.

Approximately three weeks after the patient's admission bronchoscopy was performed and half a peanut kernel was found surrounded by mucosal swelling in the left main bronchus. This was removed, but it was suspected at the time that it was not complete.

A subsequent X-ray examination showed a small area of atelectasis in the left posterior basal segment. This soon cleared and the child was completely free from symptoms three weeks after the bronchoscopy.

Case II.

A boy of three years and nine months presented on December 28, 1948, with a history of an irritating cough of approximately six weeks' duration. The cough was the only symptom and its onset had apparently been gradual. There was no obvious respiratory distress and no abnormal dyspnœa had been noticed with exercise. Physical examination revealed scattered râles and rhonchi at both lung bases. The percussion note was normal on both sides. The child was apyrexial.

Fluoroscopic examination on December 29 showed changes almost identical with those of Case I. The mediastinum and heart moved towards the right on expiration and returned to the mid-line on full inspiration, that is to say, the left lung was in a condition of fixed inflation. The diagnosis given was that of "valvular block of the left main bronchus, probably due to a radiotranslucent foreign body". The appearances on expiration and on full inspiration are shown in Figures III and IV.

At bronchoscopic examination shortly afterwards a half peanut was found embedded in the left main bronchus and surrounded by granulations. It was removed whole. The child was discharged quite well in five days' time, and has remained so.

Comment.

Of interest in these two similar cases is the fact that in both the diagnosis may easily have been overlooked on the evidence of a single film taken at full inspiration. Valvular block leading to hyperinflation is the commonest finding early in cases of bronchial obstruction in children, and the single film at full inspiration may appear quite normal or show only slight overinflation of the affected lung or lobe. Fluoroscopy overcomes this difficulty and should not be omitted in the examination of young children. An additional film on expiration would give most of the information obtained by fluoroscopy, but, especially in children a little younger than these, it may be very difficult to obtain films at a chosen stage of respiration. The possibility of an intrabronchial foreign body should always be borne in mind when young children present with vague respiratory symptoms or suspected pneumonia, and prompt action may be necessary to avoid unpleasant late effects.

The number of cases previously reported indicates that peanut kernels are peculiarly liable to be inhaled and retained, while the reactive changes they set up may make their complete removal difficult. It seems that there are considerable dangers in allowing young children this delicacy.

Acknowledgements.

Thanks are due to the superintendent of the Western Suburbs Hospital and to Dr. Eric Traill for permission to publish their respective cases, and to Dr. Milton Coutts for supplying the details of the two bronchoscopic examinations.

SIMULTANEOUS PRIMARY CARCINOMA OF THE ŒSOPHAGUS AND STOMACH.

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WHILST adenocarcinoma of the stomach and squamous carcinoma of the œsophagus are very common, their simultaneous occurrence in one person appears to be very unusual. Raven,[3] reporting 277 cases of œsophageal carcinoma, stated that, on account of the age incidence of this disease, lesions of other organs are often present, and he mentioned that in the stomach and duodenum, for instance, there may be scarring and ulceration, both active and healed, and benign tumours. However, there was no instance of coexistent primary carcinoma of other organs in this series.

It is well known that squamous carcinoma of the lower part of the œsophagus may spread to the stomach directly or via the paracardiac lymph nodes, so that the macroscopic appearance of the tumour in the stomach may simulate that of an independent primary growth.[1] Willis[4] points out similarly that the reverse may also happen. However, microscopic examination will reveal the true state of affairs.

There now follows a case report of a patient who had a coexisting primary adenocarcinoma of the lesser curvature of the stomach and a squamous carcinoma of the middle third of the œsophagus.

Clinical Record.

The patient, a well-nourished woman, aged seventy-six years, was admitted to the Royal North Shore Hospital with a six weeks' history of retrosternal pain on swallowing solid food. During that time she had increasing difficulty in swallowing food, whose passage seemed to the patient to be obstructed half-way down the sternum. When she was examined on her admission to hospital, solid food was immediately regurgitated after being swallowed. Liquids, including milk, were still able to be swallowed, though with some pain. No actual loss of appetite had occurred during this time, and the patient did not appear to have lost much weight. There were no symptoms relevant to other systems.

Physical examination revealed no abnormality save for a resting blood pressure of 200 millimetres of mercury, systolic, and 120 millimetres, diastolic. Urine examination gave normal findings and the blood picture was normal.

Radiological investigation revealed an almost complete obstruction of the œsophagus just below the aortic arch. The lumen of the œsophagus was dilated above the obstruction. The mediastinal shadow was normal.

After pre-operative treatment of the patient with a liquid Varco diet, operation was performed, the technique as described by Wookey[5] being used. The abdomen was first explored through a transverse upper left rectus incision to make sure there was no infra-diaphragmatic spread of the œsophageal neoplasm. This was confirmed; but, surprisingly, a rounded, hard mass, one inch in diameter, was found in the stomach wall half-way along the lesser curvature. It has not involved the serosa, but adjacent to it was an enlarged, hard, freely movable left gastric lymph

ILLUSTRATIONS TO THE ARTICLE BY DR. THOMAS F. ROSE.

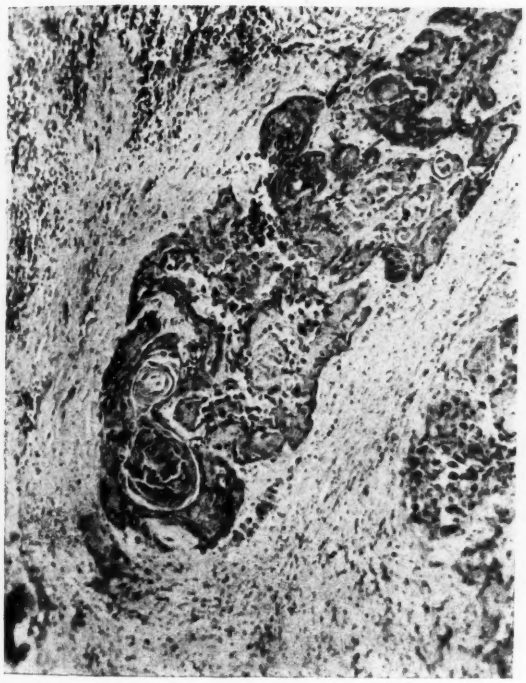

FIGURE II.
Microscopic picture of the squamous carcinoma of the œsophagus (× 12).

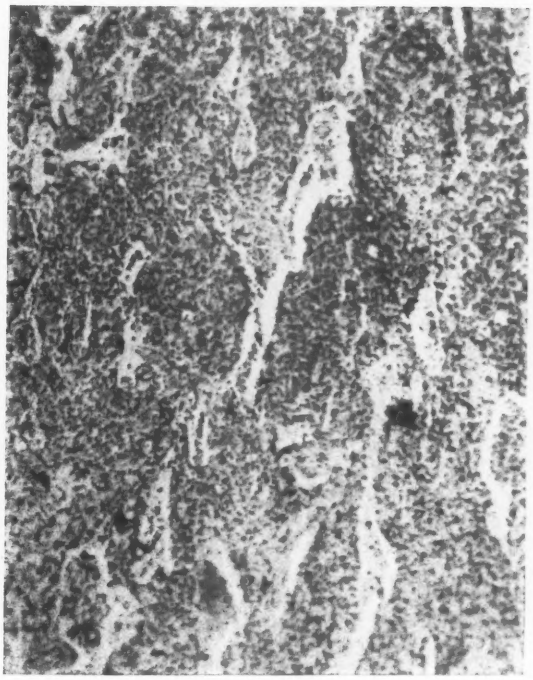

FIGURE III.
Microscopic picture of the adenocarcinoma of the stomach.

ILLUSTRATIONS TO THE ARTICLE BY DR. C. DUNCAN AND DR. W. R. PITNEY.

FIGURE I.
Endometriosis in muscle of sigmoid colon. (× 200.)

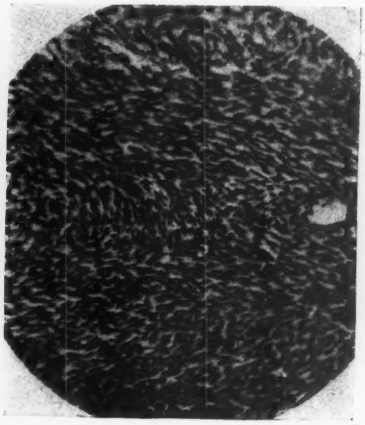

FIGURE V.
Case II. Stromal tissue. (× 200.)

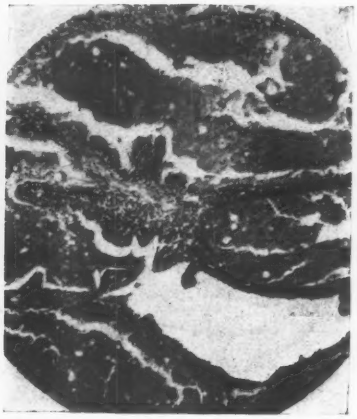

FIGURE VI.
Case II. Glandular tissue with hæmorrhagic débris. (× 84.)

ILLUSTRATIONS TO THE ARTICLE BY DR. BRIAN E. FRECKER.

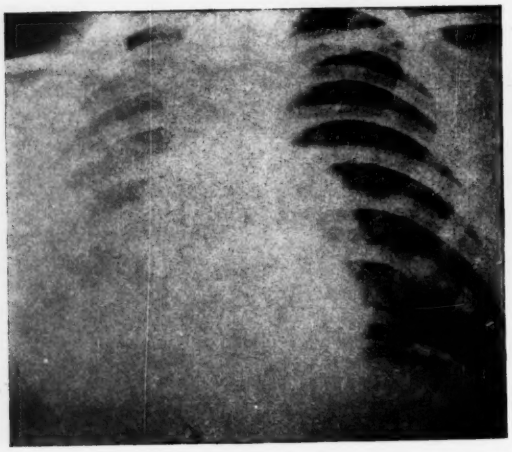

FIGURE I.
Case 1: expiration.

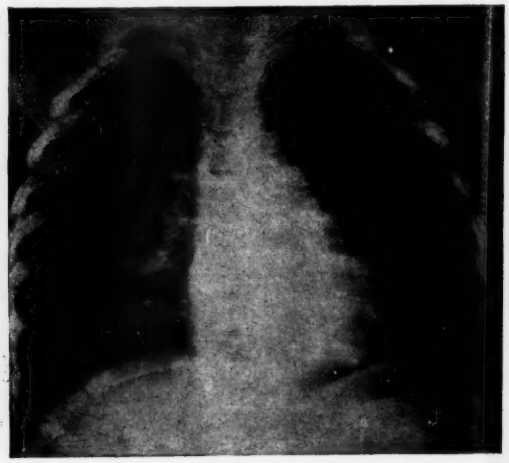

FIGURE II.
Case I: full inspiration.

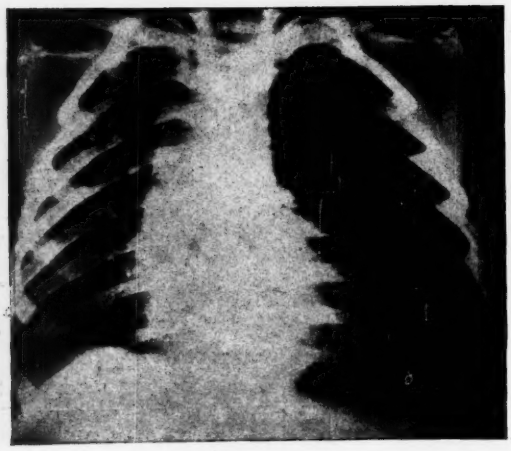

FIGURE III.
Case II: expiration.

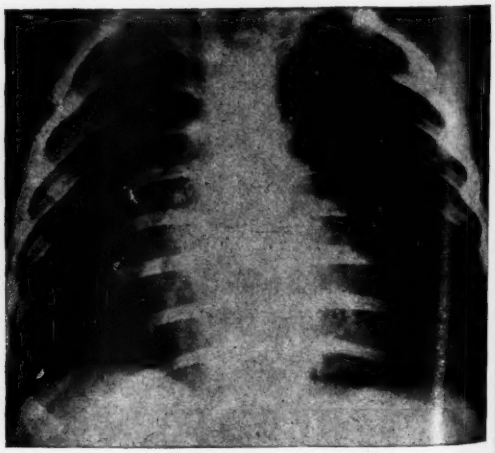

FIGURE IV.
Case II: full inspiration.

node. The rest of the stomach appeared normal and no other nodes seemed to be affected.

The incision was then prolonged laterally between the seventh and eighth ribs, the eighth costal cartilege being divided and the thoracic cavity thus opened. The œsophageal neoplasm was palpated and found to be in the middle third of the œsophagus, its upper limit being about two inches below the arch of the aorta. It was freely movable and had not spread beyond the œsophageal wall. The posterior mediastinal lymph nodes were not palpable. The left leaf of the diaphragm was split to the œsophageal opening and the stomach and œsophagus to well above the growth were resected, intestinal continuity being established by intrathoracic anastomosis of bowel and œsophagus. This was able to be done without the necessity of dislocating the proximal end of the œsophagus to the left of the aortic arch.[3]

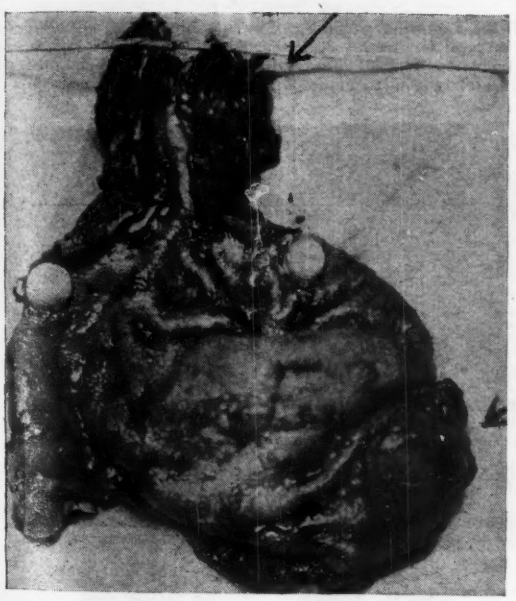

FIGURE I.

The specimen has been split along the lesser curvature of the stomach and the right border of the œsophagus. It is pinned out by drawing pins and arrows point to the neoplasms. (Unfortunately the specimen shrank in formalin before it was photographed.)

The patient did well for three days, when she died of heart failure. Unfortunately, permission for an autopsy was not obtained.

Pathological Examination.

A pathological examination of the specimen was made. Macroscopic examination (Figure I) revealed a hard, ulcerated growth in the middle third of the œsophagus. It involved the mucosa and muscle coat. It had almost completely obliterated the lumen. Half-way down the lesser curvature of the stomach there was also a hard, ulcerated mass involving all coats of the stomach except the serosa. Adjacent to it was a hard enlarged lymph node. The stomach and œsophagus between the growths appeared to be normal.

Microscopic examination of sections of the œsophageal neoplasm revealed a well-differentiated squamous carcinoma with cell nests (Figure II). Sections of the stomach growth were prepared, and a fairly anaplastic adenocarcinoma was revealed with an attempt at formation of acini (Figure III). The same picture was seen in the lymph node whose structure was replaced by the metastatic adenocarcinoma.

Summary.

A case report is presented of a patient with simultaneous primary adenocarcinoma of the stomach and squamous carcinoma of the œsophagus.

Acknowledgements.

I wish to thank the general medical superintendent of the Royal North Shore Hospital, Dr. W. Freeborn, for permission to record this case. I am indebted to Dr. C. S. Graham, morbid anatomist to the Institute of Medical Research, Royal North Shore Hospital, for the pathological investigation.

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(3) R. W. Raven: "Carcinoma of the Œsophagus: A Clinico-pathological Study", *The British Journal of Surgery*, Volume XXXVI, 1948, page 70.

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ENDOMETRIAL TUMOURS IN THE EXTREMITIES.

By C. DUNCAN and W. R. PITNEY,

Hobart.

ENDOMETRIOSIS, both uterine and extrauterine, is a relatively common condition.

Many theories have been advanced to explain the presence of endometrium in abnormal places, and no one theory seems to cover all cases. In the majority, it is likely that the tissue in endometriosis arises from the uterine mucosa, either by invasion of lymphatics, or from "spill" during menstruation. According to Goodall,[1] in the former case the ectopic endometrial tissue, being derived from the deeper layers of the musoca, is not responsive to the changes seen in the menstrual cycle, whereas in the latter it is sensitive; thus the formation and rupture of blood cysts is common, symptoms thereby being aggravated during menstruation.

For the endometrial "parasite" to survive and establish itself, theory suggests that the "soil", whether it be ovary, ligaments or bowel, must be in a suitable condition. This may be brought about by a number of causes, but hyperœstrinism, or at least sustained œstrinism, is the most important.

Endometriosis is frequently found in the ovaries and adjacent organs, and becomes progressively less common in the more distant organs.

Endometriosis, like fibroid tumours in the uterus, should not be regarded as a disease, but as one of the results of ovarian imbalance. As in fibroid tumours, too, the condition is seldom found associated with pregnancy, especially normal pregnancy. Goodall,[2] in fact, suggests that it is a product of our civilization, in that late marriages and still later conceptions are the rule.

Endometriosis resembles malignant disease in several ways. It invades normal tissues, is not normally encapsulated, spreads by local growth or implants (particularly after rupture of a cyst), and may metastasize by lymphatics and occasionally by veins (see Frank[4]). It differs, however, in not digesting the tissues of the host, in not causing cachexia, in growing more slowly, and in remaining dependent on ovarian function (ceasing to grow and slowly disappearing after the menopause or after castration by surgery or irradiation). Endometriosis might perhaps be called the "mistletoe of medicine".

Two cases reported by Goodall[3] were unusual, in that the tumours, though very radio-sensitive at first, finally some ten years later became insensitive, and caused a "frozen" pelvis with death from uræmia. Stromal metastases were present in the lungs.

Usually, endometrial masses consist of both glands and stroma, but either may overshadow the other. Figure I shows a typical example in hypertrophied muscle of the sigmoid colon, and the close association between stroma and muscle is demonstrated.

The frequency of endometriosis is difficult to assess; much depends on the skill of the gynæcologist.

Masson,[5] in a review of 2686 individual cases in the Mayo Clinic from 1923 to 1945, gives the following figures:

Uterus	1852 cases
Ovary	904 cases
Pelvic peritoneum	511 cases
Sigmoid colon	360 cases
Fallopian tube	200 cases
Uterine ligaments	192 cases
Recto-vaginal septum	67 cases
Bladder	62 cases
Cervix	45 cases
Vaginal wall	44 cases
Abdominal wall	37 cases
Small intestine	35 cases
Cæcum	18 cases
Appendix	15 cases
Umbilicus	11 cases
Ureter	8 cases
Femoral hernia	2 cases
Labia	1 case
Vesico-vaginal septum	1 case

It will be seen that the appendix, the umbilicus, the ureters and the labia are rare sites, and that only two cases occurred in association with a femoral hernia.

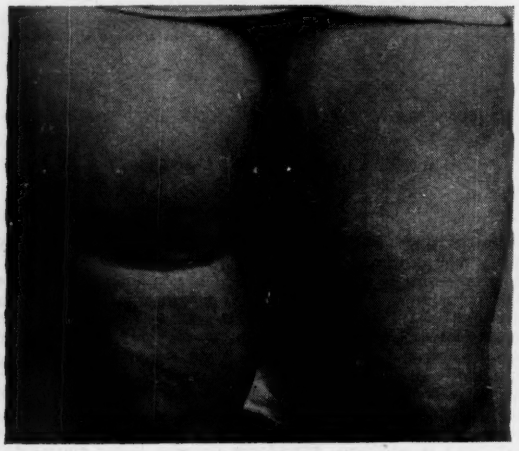

FIGURE II.
Case II. Tumour of the thigh, front view.

Marshall[6] reported a benign endometrial mass measuring 10 millimetres by 13 millimetres deep in the substance of a kidney in a woman, aged forty years. He concluded that the condition resulted from inclusion of early Müllerian cells in the metanephros.

Endometrial tumours of the extremities are very rare indeed, and few examples could be located in the literature. Rousselin[7] states that Dr. Zahedi found six cases in the groin in 165 examples of "extraperitoneal endometriosis". He then describes a woman, aged forty-two years, who developed a hard, craggy, mobile tumour the size of a pigeon's egg in the scar six years after operation for femoral hernia. The lump was painful during and just after menstruation, walking being almost impossible. No other abnormality was found. Examination of sections revealed endometriosis. Navratil[8] saw a woman, aged twenty-five years, with an endometrial tumour of the arm in the body of the *extensor carpi radialis* muscle. Professor da Costa[9] describes a woman, aged forty-three years, who, since the age of twenty-five years, had painful nodules in the inner aspect of the thighs, which became red before menstruation and finally bled. Nodules showed scar-like changes after menstruation preceding new ones which appeared soon afterwards. She had had similar formations seven years before in the lower mammary grooves on both sides, in the pectoral regions, and in the axillary plicæ. Da Costa also refers to two other cases of endometriosis, one in the thigh and one in the hand. Schlicke[10] describes in detail ectopic endometrial tissue in the posterior portion of the thigh in a Philippine army nurse. Here, the history of periodic swelling, pain and tenderness of the tumour concurrent with menstruation suggested susceptibility to endocrine influences. Moloney[11] describes three cases in the groin.

To these we wish to add two more cases.

Case I.

A.B., a female patient, aged forty-three years, noticed a small lump in the right groin which had been present for three years. She complained of a "stinging" pain in that area, but none of her symptoms was influenced by the menstrual cycle. Her menses were normal and regular. She had two healthy children.

At operation an irregular mass of tissue was removed from the external inguinal ring. No other abnormality was found. The tumour was firm and small, being less than one centimetre in its greatest diameter.

On histological examination portion of the tissue resembled normal endometrium as in Figure I, while in other areas the glandular acini were dilated by menstrual débris.

Case II.

D.W., a married woman, aged twenty-nine years, was admitted to hospital on August 3, 1948. About eight years before, she had noticed a small lump in her right thigh which appeared soon after the birth of her first child. The lump increased in size during each of her four successive pregnancies, but was not influenced by menstruation. On examination of the patient, a firm, tender swelling was present in the femoral triangle, the size of a coconut (Figures II and III). It was freely mobile on the deep structures from side to side but not up and down. X-ray examination showed a portion of the tumour one inch in diameter to be ossified.

FIGURE III.
Case II. Tumour of the thigh, lateral view.

On September 2, 1948, a large, firm, fleshy tumour, which appeared to be in Hunter's canal, was removed (Figure IV). The tumour was roughly oval and measured 10 centimetres by 7·0 centimetres by 7·0 centimetres. It was surrounded by a capsule which had small pieces of muscle adherent on the outside. The femoral vessels were stretched over the posterior wall and had to be removed. On section, the tumour was firm and grey, with irregular areas of cyst formation, hæmorrhage and ossification.

Histologically, this specimen was most puzzling. The first sections were cut quickly, as the question of amputation was put by the surgeon. They showed almost pure stroma as in Figure V, and a diagnosis of fibroma was made. Dr. A. H. Tebbutt, however, suggested endometriosis—a diagnosis which proved correct when further sections were cut. The opinion of Dr. E. Novak was sought, and he reported as follows:

> . . . Had I not known the source of the lesion I believe I would have decided very promptly that it represents a so-called malignant stromatosis, which is essentially a form of endometrial sarcoma. Endometrium has been found, as you probably know, in both the upper and lower extremities, although the total of cases of this sort is very small, probably not over four or five. So far as I know no instances of neoplastic change in these aberrant areas has ever been noted, and yet the histology of this particular lesion is exactly the same. The stromal

cells are rather markedly anaplastic, with a good many mitoses. I suppose that most general pathologists would diagnose the lesion simply as a small spindle cell sarcoma, although they might be put to it to explain the glands, since the latter seem typically endometrial, and the lesion otherwise conforms with the so-called malignant stromatosis.

Dr. R. A. Willis also favoured us with his opinion, as follows:

The specimen is certainly an unusual one, but I feel sure that it is of endometrial nature, and from its cellularity I think it must be regarded as a true neoplasm and not mere endometriosis. I think it is related to the so-called sarcomas of the endometrium, which in turn are related to the mixed tumours of the uterus. The predominant tissue in your specimen is undifferentiated stroma which, however, merges with the epithelium lining the spaces. From this point of view, it is an extremely interesting specimen, showing that endometrial stroma and epithelium have a common derivation, and thus throwing light on the peculiar mixtures of epithelium and the variety of non-epithelial tissues which are seen in mixed tumours, the latter showing differentiation of the stroma into mucoid tissue, cartilage, smooth muscle, and striated muscle also. This view, that the endometrium is a plastic tissue with very diverse potencies, is not an orthodox one, but I feel sure that it is the only explanation of the mixed tumours and of transitions such as are clearly shown in your specimen.

As to prognosis, the long history and the uniform structure of the growth would suggest that it is relatively benign and this accords with the scarcity of mitotic figures. The slight infiltration of the muscle at one point is not very significant, I think, since infiltration is of course present also in simple endometriosis. Many of the "endometrial sarcomas" (other than the highly malignant mixed tumours) which have been reported, have proved to be relatively benign and of long duration.

FIGURE IV.

Case II. Macroscopic specimen, natural size. F.V. = femoral vessels.

There was no decidual reaction of the tissues in any of the areas of which sections were examined. D.W. was fully examined and her chest and spine were radiologically investigated. No abnormality was found. She left hospital on September 15, 1948, and remained well during the next six months. She was delivered of a full-time normal child on March 30, 1949. In May, 1949, the thigh tumour recurred locally and was removed surgically. This tumour consisted of pure stroma and was rich in mitotic figures. Deep X-ray treatment to the leg with removal or destruction of both ovaries was then recommended.

Discussion.

Case I is straightforward and is reported only because of its rarity.

Case II is most unusual and perhaps unique. The most interesting points are the position of the tumour, the multiple tissue components (stromal, glandular (Figure VI), mucoid and osseous), and the infiltration of voluntary muscle by the stroma.

It is difficult to decide whether, as suggested by Willis, endometrium is a plastic tissue and was capable of forming the other tissues described, or whether the tumour was a teratoma from the start with endometrium predominating. If the tumour is only endometrial, whence did it come? Retrograde embolism seems the only possible answer.

Summary.

1. Reference is made to some aspects of the pathology and aetiology of endometriosis.

2. The relative frequency of endometriosis in various sites is given.

3. Two cases of endometrial tumours in the leg are added to the literature.

Acknowledgements.

We wish to thank Dr. J. B. G. Muir and Dr. P. Braithwaite for the case histories, and Dr. E. Novak, Dr. A. H. Tebbutt and Dr. R. A. Willis for their reports.

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SOLITARY CYST OF THE KIDNEY.

By V. S. HOWARTH,

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WITH regard to solitary cystic disease of the kidney, Hinman (1935) states: "The lack of urinary symptoms and signs and the finding of a normal pyelogram, unless the cyst perchance causes some pressure deformity, renders

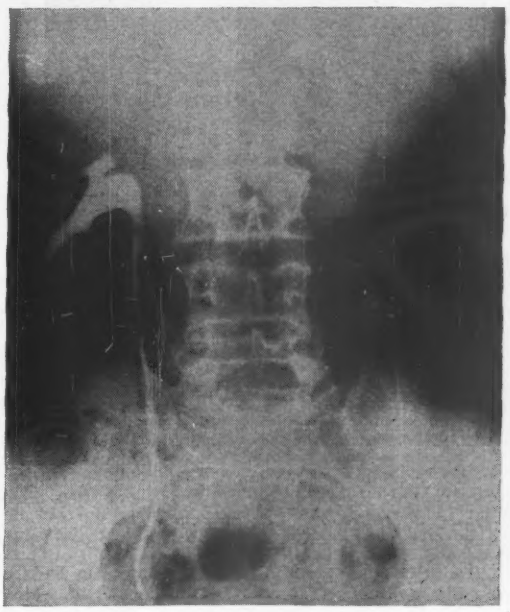

FIGURE I.

diagnosis very difficult." He also affirms that hæmaturia is present in less than 10% of these cases. The recent occurrence of an example of this disease presenting the clinical features of painless hæmaturia, renal colic and a

palpable tumour, rendering a clinical and operative diagnosis of renal neoplasm difficult to exclude, therefore warrants a record.

Clinical Record.

A man, aged fifty-seven years, was well until ten months before the time of examination. He had then noticed a sudden sharp pain, which he referred to the right costo-vertebral angle; it lasted for two hours and then ceased, leaving no residual pain or discomfort. Fourteen days later he had noticed the onset of painless hæmaturia, which lasted for two days, the blood being uniformly mixed with the urine and no clots being present. He had then been quite well for eight months, at the end of which a second attack of pain was experienced; the pain on this occasion was severe and radiated from the right costo-vertebral angle to the right loin and groin. The pain had persisted with remissions and exacerbations for one week. During the week prior to examination he had noticed a constant dull ache in the right costo-vertebral angle and right groin. He had not noticed any loss of weight.

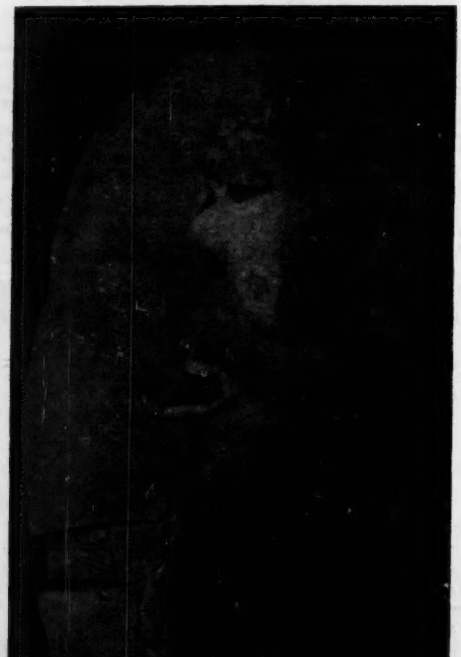

FIGURE II.

The right kidney contains a simple cyst arising in the substance of its lower pole. The cyst is 4·5 centimetres in diameter and has caused elongation and compression of the lower calyx. It is to be noted that only a very small portion of the circumference of the cyst presents on the surface of the kidney.

Clinical examination disclosed a healthy male. A smooth round tumour was palpable in the right hypochondrium, which had all the characteristics presented by a renal tumour. A clinical diagnosis was made of a right renal neoplasm. An ascending urogram was obtained, and this substantiated the clinical diagnosis (Figure I).

Right nephrectomy was performed. It was impossible at operation to differentiate the condition present from a renal neoplasm. The specimen is shown in Figure II.

Summary.

1. A simple renal cyst with clinical features closely resembling those presented by a renal neoplasm is reported.
2. The pyelogram presented a picture similar to that met with in some cases of renal tumour.
3. It may be impossible at operation to differentiate a simple renal cyst from a renal neoplasm.

Acknowledgements.

I am indebted to Dr. J. S. W. Laidley for permission to publish this case record, and to the department of medical artistry of the University of Sydney for the photograph.

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Reviews.

HOW TO BECOME A DOCTOR.

"HOW TO BECOME A DOCTOR", by George R. Moon, is described on the title page as "A complete guide to the study of medicine, dentistry, pharmacy, veterinarian medicine, occupational therapy, chiropody and foot surgery, optometry, hospital administration, medical illustration, and the sciences".[1] After this ambitious subtitle, one is pleasantly surprised that, for the purpose for which it is intended, this aim has been well fulfilled. That purpose is to provide the American high school or college student with a survey of the requirements for entering into the study of the medical and allied professions in the United States, and with sound and accurate information regarding many important but frequently forgotten matters. The admission requirements of every medical school in the United States are listed, and the general standard of advice to prospective students is very good. Of particular interest are the chapters dealing with admission applications, interviews, and the special problems of the medical student, but these can apply only to the United States.

The book is of interest only to a limited class of readers in Australia, and will serve as a useful and brief reference book for the information of persons interested in medical education overseas.

ILLUSTRATIONS OF SURGICAL TREATMENT.

THE third edition of "Illustrations of Surgical Treatment" by Eric L. Farquharson maintains the high standard set by previous editions.[2] The text has been revised and several new ideas on treatment have been incorporated, supported by fresh illustrations. The subject matter throughout is essentially practical and the author has a flair for simplicity of description, the most important points being emphasized by the use of different type.

As before, the first part deals with intravenous saline infusion and transfusion of blood and protein fluids. Com-

[1] "How to Become a Doctor: A Complete Guide to the Study of Medicine, Dentistry, Pharmacy, Veterinarian Medicine, Occupational Therapy, Chiropody and Foot Surgery, Optometry, Hospital Administration, Medical Illustration, and the Sciences", by George R. Moon, A.B., M.A.; 1949. Philadelphia and Toronto: The Blakiston Company. 8¼" × 5¼", pp. 148. Price: $2.00.

[2] "Illustrations of Surgical Treatment: Instruments and Appliances", by Eric L. Farquharson, M.D., F.R.C.S. (Edinburgh), F.R.C.S. (England), with a foreword by the late Sir John Fraser, Bt., K.C.V.O., M.C., M.D., Ch.M., F.R.C.S. (Edinburgh); Third Edition; 1949. Edinburgh: E. and S. Livingstone, Limited. 9½" × 5½", pp. 408, with 61 illustrations. Price: 25s.

patibility tests, the Rh factor, methods of administration of blood and other fluids and reactions are adequately described.

The main body of the book deals with the commoner fractures and dislocations, and other orthopædic conditions such as tuberculous disease of the spine and hip joint, congenital dislocation of the hip and foot deformities.

In describing methods of reduction and maintenance of position of fractures, the author presents detailed descriptions illustrated by photographs and line drawings.

Among the outstanding sections are those dealing with injuries about the wrist, fractures of the spine and femoral shaft fractures.

A particularly clear description of Colles's fracture is given in which no detail is omitted, and the practitioner following the advice here will have few bad results. The author's own splint for navicular fractures is described. The various methods of reduction and immobilization for fractures of the femoral shaft are well dealt with and his own method of using a suspended plaster cast combined with traction has obviously been evolved from a wealth of experience.

Details of minor but nevertheless important points, such as the correct method of fitting walking irons, strapping for ankle sprains *et cetera*, are clearly given with lavish use of illustrations.

The final part consists of an appendix illustrating instruments and appliances, which should prove useful to the student.

Farquharson's book has already proved its worth and should find a place in the library of the surgeon, of the practitioner dealing with fractures, and of the student.

CRIMINAL CASE HISTORIES.

THE two large volumes under review are numbers three and four of Dr. Ben Karpman's "Case Studies in the Psychopathology of Crime", and represent a continuation of the case material he has already presented in the earlier volumes.[1] The eight criminals recorded in these volumes were all murderers, two of whom had committed burglary in addition. The writer is a well-known psychotherapist at St. Elizabeth's Hospital in Washington, D.C., and the material was collected from those who had been patients in the criminal section of the hospital. Each volume is prefaced by the same picture reproduced in colours of "Christ After Flagellation", having been painted by a criminal patient while in hospital, and meant to symbolize much of the prevailing attitude of prisoners to their incarceration. The painting is analysed from the artistic point of view by a well-known art critic and curator of the National Gallery at Washington. Each volume is further supplied by a general preface couched in exactly the same terms and setting out the method of the investigations. This represents an extremely useful key to the books and contains the pith of the writer's conception of the ætiology and psychopathology of crime.

The eight case histories presented in these two volumes are as complete as possible; and it would seem safe to state that seldom, if ever, have such detailed investigations of individual criminals been presented. Interpretative comments have been deliberately omitted. Each case stands stark in the light of common day. Nothing is glossed over. Nothing is withheld. The unpalatable is paraded before the reader with an almost merciless honesty. Generous impulses vie with revolting practices and mean-spirited actions go with greed and the swollen instincts of lust. Dirty linen from the intimate family lives of these criminals is set upon visual display. There are glimpses into many a thieves' kitchen. All the petty deceits of youth, the aggressions and frustrations of the poorly conditioned, the futile punishments and inane threats, the pleadings of bewildered parents, the inevitable return to the crooked pathways of vice that lead almost inexorably to adult criminal behaviour and its frequent association with alcoholic and sexual debauchery, are in this Gargantuan gallery of the criminal insane.

This compilation, therefore, becomes a valuable source-book for the criminal psychiatrist—a veritable treasury of criminal trends and precipitating factors relating to the evolution of the antisocial individual whose aggression has placed him behind the bars of common justice. But, while admiring the indefatigable pursuit of detailed knowledge displayed by Dr. Ben Karpman, one wonders how many will ever wade through the torrid pages of such a voluminous history. Psychiatrists know that psychotherapy offers the only hope to the criminal. They realize, as does Karpman, that neurotics are repressed criminals and that most criminal behaviour is the result of unrepressed neurotic impulses. But when will the public and the judiciary realize these matters? Neither is ever likely to undertake the perilous adventure of reading these volumes. And if by some miracle they did it might only show them that while psychotherapy may help to rehabilitate the individual criminal, it is doubtful whether it could ever abolish crime from our social order. Crime as a reaction to social control is still the basic enigma. The light which these histories throw into many dark places serves only to illuminate further the fundamental conundrum of crime.

[1] "Case Studies in the Psychopathology of Crime: A Reference Source for Research in Criminal Material", by Ben Karpman, M.D.; Volume III, Cases 10-13; Volume IV, Cases 14-17; 1948. Washington: Medical Science Press. 10⅜" × 8⅛". Volume III, pp. 878; Volume IV, pp. 918.

NURSING.

IN "Ward Administration and Clinical Teaching", Florence Meda Gipe and Gladys Sellew have produced a book which, although written from the point of view of nurses in the United States of America, has much that is profitable for nurses in Australia.[1] It is divided into two sections which are of necessity closely connected. In the first, ward administration is discussed, mainly from the point of view of the head nurse and the supervisor. The authors are aware of the fact that "the administration of the hospital begins in the head nurse unit", and it is their aim to present simple principles to increase the efficiency of this unit. They touch on many aspects of ward management, from finance and cost accounting to service and research in nursing practice. They rightly insist that schools of nursing should exist for the sole purpose of education, and not to staff the hospital at the least possible expense; yet they also rightly insist that service to the patient is the prime function of the hospital. It is recognized that these two aspects may create tension, but it is pointed out that this is not insurmountable, as there is no inherent conflict between the needs of the patient and those of the student nurse.

The second part of this book deals with clinical teaching; and while it is not, and does not profess to be, a manual of nursing education, yet it does give some helpful discussion on this aspect of teaching, which is all too often neglected or inadequate. The value of a ward library is stressed, much space is spent on the explanation of the "pretest", and there are some excellent rating devices for both student nurses and subsidiary workers. While some of this book would seem at first sight to be of theoretical value only, yet it would be of great advantage if those who are, or should be, interested in these subjects were to study the book and put some of its recommendations into practice.

A TEXT-BOOK OF MEDICINE.

WE can heartily endorse the contention of the publishers that in its new format the ninth edition of Conybeare's "Textbook of Medicine" is much more easy to handle, for we had mentioned in a previous review that in the original format the book was growing rather too bulky to manage.[2]

While in the main the subject matter has undergone little alteration, it has been found necessary to make a good many minor revisions of the context in order to incorporate reference to some of the newer adjuncts to diagnosis and treatment.

A few of the articles, however, have been entirely rewritten, and this particularly applies to much of Dr. F. M. R. Walshe's outstanding contribution on diseases of the nervous system. At the present juncture it is of special interest that one of the sections Dr. Walshe has rewritten is that dealing with poliomyelitis, which he has, to some extent, reorientated in the light of experience gained in a recent outbreak of the disease in Great Britain. Anyone wishing to refresh his

[1] "Ward Administration and Clinical Teaching", by Florence Meda Gipe, M.S., R.N., and Gladys Sellew, Ph.D., R.N.; 1949. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 8½" × 5½", pp. 357, with 21 illustrations. Price: 32s.

[2] "Textbook of Medicine", by Various Authors, edited by Sir John Conybeare, K.B.E., M.C., D.M. (Oxon.), F.R.C.P.; Ninth Edition; 1949. Edinburgh: E. and S. Livingstone, Limited. 9¼" × 6¼", pp. 896, with 28 illustrations. Price: 30s.

knowledge on this subject will find here an admirable monograph.

It is unfortunate, as the author himself points out, that the interval that in these days elapses between the setting up of the type and publication of a book is so long that it is impossible to keep pace with modern advances in medical knowledge, so that in some of its aspects a new edition is out of date before it reaches the hands of the book-seller. Thus, in the light of our present knowledge, it seems strange to find Sir John Conybeare in his preface dismissing streptomycin as a treatment in the experimental stage and Dr. Walshe writing that symptomatic treatment only was available in the case of tuberculous meningitis.

However, from the student's point of view such omissions are of little practical moment, as it is naturally of the newest treatment that he is likely to hear most in his clinical teaching.

Typographical errors are, of course, unavoidable, but it is rather jarring to find in the article on essential hypertension Allbutt referred to as Sir Stafford Allbutt.

That, of course, is a minor matter, and we have no hesitation in endorsing our previously expressed opinion that this is one of the most outstanding short text-books of medicine available.

HISTOLOGY AND HISTOPATHOLOGY OF THE EYE.

TEXT-BOOKS on pathology of the eye are so few that the announcement of a new one is of more than usual interest. First impressions of "Histology and Histopathology of the Eye and its Adnexa", by I. G. Sommers, M.D., are unfavourable. There is no attempt to catch the eye with profuse and elaborate illustrations. The setting out of the text is unattractive and the index inadequate. Unlike a recently magnificently bound volume on a similar subject, it can scarcely be called a collector's piece. Nevertheless, it covers the field of ocular pathology in a far more comprehensive manner than any other book of recent years.

The first part on the normal histology, embryology and senescence of the eye is so sketchy that it might well have been omitted. Part two, on general pathology in relation to the eye, serves as an excellent introduction to the subject proper. This is arranged in the usual manner under anatomical headings with separate sections describing special diseases and developmental defects. Diktyoma of the ciliary body is, curiously enough, included in diseases of the uvea. Each section is followed by an extensive bibliography.

By deliberate omission from the text of the "names of the many hundreds of writers—whose opinions are so very often contradictory", the author claims to have aided the clarity of presentation. After each chapter, under the heading "Reading of source material", these opinions are summarized without comment. The discriminating reader will appreciate the objective approach of the author, who makes no attempt to conceal the present lack of accurate knowledge of pathological processes in the eye. He does this nowhere better than in the section on glaucoma, a subject popular with "armchair pathologists".

The book can be well recommended to the student of ocular pathology as well as to the general pathologist asked to give an occasional opinion on a section of the eye. The practising ophthalmologist will also find it a valuable source of reference.

THE MENTAL LIFE OF THE CHILD.

IN "The Mental Life of the Child", Dr. Gustav Hans Graber condenses his previous writings in child psychoanalysis.[2] He believes that the unconscious is the true being and that the God-like in man is often accessible only when the unconscious is revealed. His book is addressed to all preceptors, parents (especially mothers), teachers, priests, physicians and psychologists. To this end it is written in a somewhat popular vein.

The author claims that dreams are the key to the unconscious life in children. After discussing simple dreams due to internal bodily stimuli, such as the full bladder, or those in which they are simple wish fulfilments of waking life, he proceeds to an analysis of those with deeper meaning. It is significant that he prefaces his excursion into these realms of darkness with a soliloquy upon man's fallibility which has resulted in an endless series of devastating wars. To him there is only one answer. "The mighty, tabooed instincts were pushed aside. But the fettered primitive forces tugged at the chains forged from resolutions and 'good will' like caged beasts, pushed like a dammed torrent, and set themselves free, spreading devastation and destruction, like revengeful gods or furious elements."

The first case history illustrates the transformation of aggression in a three-year-old child. The girl was a veritable criminal who turned an ordered household upside down. Analysis revealed hatred for a younger sister and the mother who had supplanted her in the affections of the father.

The next case illustrates Dr. Graber's favourite theme of the reality of the infantile wish to return to the mother's womb. Paul, aged nine, was an ineffective day-dreamer. He enjoyed sleeping rather than play and had no urge to grow up. He had an extraordinary liking for lifts and closed spaces. His dreams were interpreted as disguised wishes to be in the warm safety of his uterine existence.

Many of the histories reveal a predominantly sexual basis for the abnormality. Elsa was an "impossible" child who spent her major energies in inflicting pain on others. Analysis revealed that at the age of four she saw her parents in sexual intercourse. Thenceforward she had an inordinate curiosity to know what had happened and phantasy after phantasy chased through her brain. She wished to be in the same relationship to her father and became dominated by urges of jealousy. Sadistic traits were paramount. The author through psychoanalysis was able to give her insight into her infantile fixations and obtain a successful therapy.

A more conventional story surrounds the four-year-old whose "maid pulled my hair nearly every day, and she shut me in the bathroom for hours, and then turned on our gramophone and danced. I always cried when daddy and mummy went away. I was four then. I knew she did with me whatever she like. She hit me too. She locked me in so that I shouldn't see or tell anything. She boxed my ears and kicked me too. I never said anything, because she might have been still more cruel to me." The result was a submissiveness which brought him for treatment.

The majority of the material is woven around his interpretations of sex, thoughts of incest, love and hate, anal pleasures. The author is careful to point out that the analysis does not inspire the theme; he merely records what he observes. In this respect the reader cannot doubt his veracity.

Children's thoughts and emotions have the variegated and vivid colouring of old world fairy-tales. There are gnomes and giants, evil witches and dreadful demons. Dr. Graber exploits this theme. His patients appear to be at home in fairyland and are acquainted with a wide variety of its inhabitants who are unknown to Australian children. Furthermore, his children have a verbal imagery which suggests that the author has dealt with a special type of bright child.

A perusal of the book does not solve the problem of whether generalizations based on a selected group of special children is applicable to all children. It does, however, indicate the dynamic quality which underlies the thoughts of children. The book is provocative and well worth a perusal. It highlights the need for the psychological approach to many of the problems of childhood.

[1] "Histology and Histopathology of the Eye and its Adnexa", by I. G. Sommers, M.D.; 1949. New York: Grune and Stratton. 8¼" × 5¼", pp. 804, with 69 illustrations. Price: $12.00.

[2] "The Mental Life of the Child", by Gustav Hans Graber; 1949. London: Staples Press, Limited. New York: Staples Press Incorporated. 7" × 5¼", pp. 164. Price: 8s. 6d.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Anæsthetics and the Patient", by Gordon Ostlere, M.A., M.B., B.Chir., D.A.; 1949. London: Sigma Books, Limited. 7¼" × 4¼", pp. 170, with a few illustrations. Price: 7s. 6d.

Intended for the non-medical public.

"Reports on Biological Standards: III. Methods of Biological Assay Depending on a Quantal Response"; Privy Council. Medical Research Council Special Report Series, Number 183, by J. H. Gaddum; 1933, reissued 1949. London: His Majesty's Stationery Office. 9¾" × 5¾", pp. 48. Price: 1s.

A reissued report.

"Progress in Neurology and Psychiatry: An Annual Review", edited by E. A. Spiegel, M.D.; Volume IV; 1949. New York: Grune and Stratton. 9" × 5", pp. 612. Price: $10.00.

Chapters are written by 67 individual contributors.

The Medical Journal of Australia

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All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

SCIENTIFIC BIOGRAPHY.

THE views of an eminent English physician with a sense of humour on the subject of the anamnesis (*alias* the medical history of a patient) were mentioned in these columns on April 30, 1949. A nicer complement to these views could not be desired than the ideas of an eminent American psychiatrist is Carl Binger,[1] Associate English physician was Sir Adolphe Abrahams, the American psychiatrist was Carl Binger,[1] Associate Professor of Clinical Psychiatry at Cornell University Medical School, New York. Discussing the purpose in taking a medical history, Binger suggests that it is at best a kind of stylized symbolic representation of certain human experiences, much as a map is a stylized symbolic representation of a landscape. The value of a good map will depend upon the accuracy of its scale and upon the picturing of the relationship of certain important land masses, contours and bodies of water, as well as of major man-made structures. With the aid of such a map, we can find our way around in strange country. "But", Binger points out, "if the map depicts every culvert, duck pond and hot dog stand and leaves out mountain ranges and navigable rivers, we will soon go astray if we rely upon it." Just so a good medical history will portray in the dimension of time, rather than space, the influences, past and present, that are important in the development of the patient and his disorder. Binger describes the ritual followed at the beginning of his medical career when he was taught to take the kind of standardized history "which includes everything and tells us nothing". He goes on:

> We were as persistent as a cigar-smoking detective in a movie melodrama and about as subtle. I recall asking a young woman about her weight. What was her present weight, her weight last year and her heaviest weight? When I asked her what her lightest weight was and she said "5½ pounds!" I began to get the notion that perhaps I was boring her by the third degree method.

After all, patients, especially those acutely ill, should be spared what they will regard as silly questions, if possible. Another example of a silly question is in Binger's recollection of the chief attending physician, a sober and pedestrian man, asking a little boy of eight whether the pain in his groin radiated to his penis and testicles. The child looked up bewildered and said: "What's them?" The last part of the history was the "P.I." (presumably the account of the present illness), which had "little to do with past events and less with what today we call the personality". But the histories were nothing if not complete—the greatest sin was the sin of omission—and

> they bore about as much resemblance to the true story of the patient's life as a Mercator projection does to the contours of the earth's surface.

At the other extreme is the history written by a doctor whom Binger had urged to stick to essentials instead of writing long rambling histories as was his usual custom. The history was as follows:

> Complaint: Abdominal pain.
> Past history: Kicked by a steer.
> Diagnosis: Tonsillitis.

This Binger describes as "a paradigm of the nonsequitur and an excellent caricature of many medical histories today". The point of resemblance making the caricature is presumably the *non sequitur* and not the brevity, for we cannot deny his assertion that the long history full of irrelevancies is "still the current method in most general hospitals and in many private offices, although in some" he adds a trifle cynically, "history taking is but a scanty business or is turned over to an office nurse who at the same time sizes up the patient's probable income".

The alternative to the time-worn method is what Binger terms "scientific biography", a happy phrase which provokes thought. He suggests that a change in this direction is taking place at least in some places and claims that it is largely due to the influence of "modern dynamic psychiatry". We need not quarrel with that, but another contributory factor is of particular interest. Binger points out that the establishment of the germ theory last century led to a type of "simple unitary etiologic thinking" exemplified by Koch's laws which brought great successes, but—and this is a paradox—the consequent reduction in incidence of infectious diseases has brought into relative prominence the degenerative diseases and stress reactions to which this type of thinking is far less applicable, and because of its success it is being displaced. Our aim then in taking a medical history, according to Binger, is no longer simply to find an ætiological agent and then "to discover means of exorcising it". It is rather to discover what kind of individual we are dealing with, what are his strengths and weaknesses, his assets and liabilities, his patterned reactions under stress and in what respects the bodily defences, which he mobilizes, themselves turn into malign and damaging forces; for, as Harold Wolff (quoted by Binger) has pointed out, although man's protective activities may be life-saving if they are used in emergencies to destroy the forces that threaten him, the same protective activities when used as life-long patterns may damage the very structures they were devised to protect. Much of this is psychiatrist's language, as we would expect, but it at least warrants consideration in physiological and pathological concepts as much as in psychological. To follow its trend will certainly influence the course of history taking. Binger develops his ideas of the relevant technique, but we need not go into them in detail. Important points are that "the old question and answer game", especially with a printed or fixed list of queries, does not suffice; that the right contact must be established

[1] *The American Journal of Medicine*, June, 1949.

and maintained between doctor and patient; that what matters most is the patient's *persona*, the front he presents to the world and not his appearance in a hospital bed; and that every opportunity must be taken to observe and deduce the essential points which are not, perhaps cannot be, expressed by the patient. The aim is to obtain a knowledge of the person as well as of his illness, "because in most instances, perhaps in all, they are inextricably related".

It is interesting to note that not once in his paper does Binger use the blessed word psychosomatic, though that is undoubtedly the concept at the back of his mind. He accepts that concept as basic in the approach to any patient and appears to offer little encouragement to those who would speak of psychosomatic medicine as if it was a specialized field. However, his thoughts on the present-day inadequacy of simple unitary ætiological thinking may cast some light on the development of this curious specialty. Perhaps the present phase is just a stage in thought between the idea of fixed relationship between specific cause and effect and the clear concept that all medicine is psychosomatic medicine. In practice the best type of doctor has long followed the last-mentioned concept, but it is very doubtful that he ever formulated it in speech or thought. The attempt at definition has led it temporarily into what could be a blind alley, but we must not allow this developing idea to become immobilized, much less despised. The whole respective concepts of the patient in relation to disease and of disease itself are undergoing profound changes. Binger points out that if the young medical student still looks upon a patient as a case of this or that, it is our fault because we still believe in "a kind of Platonic abstraction called disease as if it existed outside the body of the sufferer". We may compare with this the views expressed by H. P. Himsworth,[1] Professor of Medicine in the University of London, in a paper on the syndrome of *diabetes mellitus*. Himsworth, taking as his text the statement that "the history of modern knowledge is concerned in no small degree with man's attempt to escape from his previous concepts", asserts that we are at present witnessing a liberation of medical thought by the substitution of syndromes for "disease entities" as the units of illness. Implicit in the concept of a disease entity, he states, is the idea that any particular illness has a specific cause, which, though its action in the body may be modified by circumstances, is an essential and invariable prerequisite for the development of the illness in question. The syndrome, on the other hand, "has its philosophical basis not in specific disease factors but in a chain of physiological processes, interference with which at any point produces the same impairment of bodily function". The same syndrome may thus arise from many different causes. If we accept this point of view the patient becomes the essential unit in the whole clinical and pathological story, and the outside influences, organismal, physical, chemical or emotional, though important are subsidiary. Every patient seeking advice presents an individual problem, straightforward in some cases, complex in others, and the "cut and dried" approach will not do. Early editions of Osler's "Principles and Practice of Medicine" began with a section on specific plasm had displaced the submucosa and had penetrated to, but not into, the laryngeal cartilages. It was later learned infectious diseases, the article on tuberculosis featuring the parable of the sower—a foreshadowing, albeit incomplete, of the present trend of thought; recent editions begin with an article on psychosomatic medicine. The constant repetition of the statement that good doctors have always in practice known this approach to their patients tends to become boring, and it might well be asked why, if this is a fact, there is the present-day striving to formulate and press home the principles involved. May it not be that with many other modern trends—regimentation of medical services, narrow and early specialization, undue reliance on mechanical aids and special tests—the patient is in danger of being forgotten? If we are to keep a right perspective in the approach to a patient, Binger's point of view might well be pondered, that for the wise doctor today the obtaining of a medical history has become primarily a personal voyage of discovery into unknown territory, in which doctor and patient are joined in a common enterprise.

[1] *The Lancet*, March 19, 1949.

Current Comment.

SIMULATED HOMICIDE.

A NICE example of the fallacious conclusions which can result from a combination of circumstantial evidence and unsatisfactory post-mortem medical examination has been reported by Joseph W. Spelman.[1] The story was that early one morning, the police were notified that an apparently intoxicated Negro man, accompanied by two women, had been seen carrying through the streets a carelessly wrapped parcel, which appeared to contain the body of a dead child. A passer-by reported overhearing the trio discussing the best means of disposing of the corpse. On arrival of the police, the man stated that the body was that of his son, aged sixteen months. The body was immediately taken to a mortuary by the police and the medical examiner notified. The mother of the dead child was a patient in a neighbouring hospital. The father stated that he had put the boy to bed on the sofa in the family living room at about 10 o'clock the night before. He had then gone to the apartment of one of the women, got drunk, and returned home at 2.30 a.m. to find his son dead on the floor near the sofa. After a conference between the father, his companion of the night and another woman, the three of them wrapped the body in a blanket and were proceeding along the street aimlessly when detected at 3 a.m.

At post-mortem examination the body was found to be dressed in urine-soaked daytime clothing. The shoes had been placed on the wrong feet and were laced, though the laces were not tied. The body was undernourished and underdeveloped. There was no external evidence of recent trauma. Incision of the body revealed marginal pulmonary emphysema and occasional subpleural petechiæ. Under the scalp a large transverse linear fracture of the right parietal bone was found, covered by a fresh hæmatoma. A scanty epidural hæmorrhagic extravasation surrounded the fracture. There were a few superficial petechiæ throughout the cerebral cortex. If, as Spelman points out, the autopsy had been concluded at this point, there would have been a strong presumption that the child had died as a result of a fractured skull with associated cerebral damage, and this with the peculiar circumstances of the story might well have led to a charge of homicide. However, the examination of the body was continued, and the larynx was found to be completely obstructed by multiple papillomatous tumours. These soft, friable, polypoid masses had obliterated the vocal cords and extended from the ventricular ligaments to the inferior margin of the cricoid cartilage; firmly attached to the circumference, the neo-

[1] *American Journal of Clinical Pathology*, June, 1949.

plasm had displaced the submucosa and had penetrated to, but not into, the laryngeal cartilages. It was later learned that the child had been examined in a children's clinic at the age of ten months because of hoarseness, muffled voice and loss of weight, and again at the age of thirteen months when increase of weight was noted, but nothing was recorded regarding the muffled voice and hoarseness; the larynx had never been directly examined. The final inference was that the child at the time of death had an upper respiratory tract infection, and that the resultant œdema, hyperæmia and exudation, superimposed on pre-existing partial laryngeal obstruction, brought about sudden occlusion. The fracture of the skull could be explained as having been sustained during the agonal period, either from a fall from the sofa or from trauma received during a terminal asphyxial convulsion. The father and his companions presumably became afraid and, being fuddled, acted foolishly, so making the circumstantial evidence many times more damning. A better illustration of the importance of complete and thorough medico-legal autopsies would be hard to find.

VERTIGO.

VERTIGO is one of the subjects which lends itself to a symposium, as it is a symptom without objective signs, but one which is so disabling that it is almost certain that the patient will seek advice. This statement is in itself a partial definition of the sensation, or at least its degree, and defines that attitude of the internist to it in a symposium recently published.[1] Mark Aisner, representing the general physician, points out that the patient is aware of some difficulty in the postural mechanism, is insecure, especially when upright, and suffers from a sense of rotation which is implied in the name vertigo. He goes on to remark that a multitude of milder symptoms, such as dizziness or faintness, are much commoner than true vertigo, and unfortunately even in text-books on medicine often masquerade as true vertigo. Aisner admits the term in some of the milder attacks of the hypertensive patient, but otherwise confines it to pure otological and neurological entities, in addition to the genuine types seen in leuchæmia and in drug intoxication. In leuchæmia an infiltration of the end organ may occur, while of drugs the most important today is streptomycin.

Others who contributed to this symposium represented neurology and otology. D. E. Denny Brown from the neurologist's point of view regards the distinguishing characteristic of true vertigo as the consistency of the sense of apparent movement, which occurs in clear-cut attacks, and is made worse by any movement. Like the physician, he agrees that tinnitus and deafness are usual accompaniments. He emphasizes that the most important guide to the neurologist is the form of the attack, which largely determines the anatomical diagnosis. Labyrinthine vertigo he classes as paroxysmal, usually sudden of onset, and brief of duration, though subsiding more gradually than it came. Naturally the neurologist is specially interested in varieties due to lesions of the brain stem.

Joseph J. Fischer discusses the otological aspects. He comments specially on the dependence of the diagnostician on the capacity of the patient to describe what he feels, but points out that confusion is not only possible from this cause, but is also produced by the careless definitions implicit in some of the descriptions in the literature. Some of these are based on true physiological processes, others on psychological considerations. After the usual presentation of the otologist's procedure and findings, Fischer remarks that while vertigo can easily be understood as a symptom of inflammatory, degenerative, toxic and traumatic conditions, it is not always correspondingly easy to understand it in vascular disturbances.

It is natural that the evaluation of a symptom should be differently made by doctors whose outlook and interests are not entirely the same. Perhaps the otologist, who is more intimate with the end organ of the labyrinth as regards its anatomy, should have the most concrete ideas about its physiology. But one wonders on reading such a symposium how clearly a definition of the condition which is being discussed emerges from the accounts given. Is one to regard true vertigo as a condition which if arising from the end organ is essentially of sudden onset? And are the symptoms as related by an intelligent patient either consistent to a pattern, or else modified by certain differences which may be of diagnostic significance? In connexion with this latter, Aisner notes that the variety of vertigo associated with the toxic effects of streptomycin has some characteristics of its own. The rotary component is often absent, and the disorientation of the postural mechanism of the patient has certain distinctive features. Thus, when a sudden movement is made, actual overshooting is not uncommon, or else there may be a distinct sensation of continuation of the movement, though this has ceased. The anatomical localization of the disturbed areas has been suggested as the ventral cochlear and inferior cerebellar nuclei, though some authorities consider that the peripheral mechanism is also disturbed. The moral of this appears to be that vertigo is far from being a worked out subject. There is still confusion in what is meant by the term, and there is still room for accurate study of the anamnesis and the clinical signs and symptoms.

[1] *The New England Journal of Medicine*, July 28, 1949.

PENICILLIN AND PERIPHERAL NEURITIS.

IN 1946 L. C. Kolb and S. J. Gray[1] reported seven cases of localized peripheral neuritis with motor and sensory disturbances developing between ten and twenty-one days after the institution of intramuscular penicillin therapy. Five patients recovered completely within four months; two had residual effects. The relationship between the peripheral neuritis and the penicillin therapy was not absolutely established, but there appeared to be strong grounds for accepting it, especially as a number of other reports had appeared indicating a probable toxic effect of penicillin on central nervous tissue. Evidence of nerve compression, direct injury to the nerve or an allergic response was lacking, and it was suggested that the peripheral neuritis represented a delayed toxic reaction to penicillin. Subsequent reports have supported the idea of the toxicity of penicillin for nervous tissue in certain cases, and a further report has now appeared describing peripheral injuries apparently due to penicillin. T. R. Broadbent, G. L. Odom and B. Woodhall[2] report four cases of a peripheral nerve lesion immediately following the administration of penicillin into or about the nerve. Intense immediate pain was felt, radiating distally along the course of the affected nerve segment, with complete (but in two cases rapidly regressing) sensory loss and early motor disability that, in two cases at least, became a chronic and static disability. It is stated that the reaction was unlike any of the well known traumatic sequelæ of, for example, procaine anæsthesia. A more intense reaction perhaps occurred in the presence of a vehicle such as wax, but it is considered improbable that the effect was wholly dependent on the vehicle. In two cases the nerve was examined at operation and the condition found suggested an intraneural lesion; evidence of extraneural compression was lacking. Broadbent *et alii* state that the neuropathological changes produced experimentally in animals are now being studied. The results of these studies will be awaited with interest, as a good deal of clarification is needed. It is rather curious that only two small groups of patients with this type of condition have been reported despite the general use of penicillin under all conditions. The difference in onset between the two groups is striking and indeed confusing if we wish to regard the two groups as representing the same condition. The possibility of peripheral damage due to penicillin in certain cases is too important to ignore—possible medico-legal implications, for example, are obvious—but we do wonder why reports of it are so few.

[1] *The Journal of the American Medical Association*, October 12, 1946
[2] *Ibidem*, July 23, 1949.

Abstracts from Medical Literature.

BACTERIOLOGY AND IMMUNOLOGY.

Agglutination of Red Cells by Hæmophilus Pertussis.

J. UNGAR (*The Journal of Pathology and Bacteriology*, January, 1949) studied the agglutination of red blood corpuscles by *Hæmophilus pertussis* in an extension of the attempt to differentiate between virulent and avirulent strains. The cultures tested would agglutinate red cells only if held at a temperature of 46° C. The experiments were then carried out on washed organisms grown for twenty-four to forty-eight hours on Bordet-Gengou medium tested against 5% suspensions of washed human red cells. Results show that while all of 32 strains designated as virulent produced agglutination of red cells, none of six strains classified as avirulent did so. The author discusses various factors which affected the phenomenon of agglutination.

Electron Microscopy of Chromosomes.

SANFORD L. PALAY AND ALBERT CLAUDE (*The Journal of Experimental Medicine*, April, 1949) state that they have made an electron microscope study of salivary gland chromosomes of Drosophila larvæ. These giant chromosomes have been studied in detail with the aid of the light microscope, but the authors have gone further, and developed a method employing formvar replicas of the intimate structures. The electron microscope photographs produce negative pictures of rows of small granules or chromomeres arranged from side to side, and these make up the whole structure. In stretched-out preparations a network of filaments appears between the granules of two consecutive rows, but these are believed to be artificially produced. If such a preparation is submitted to the action of desoxyribonuclease, the filaments disappear and only amorphous material remains. There was no evidence of a limiting membrane around the rows of chromomeres, and the authors believe that the effects of enzyme action suggest that at least portion of the nucleic acid lies on the surface of the chromomere.

Virus Studies on Patients with Mumps.

EDWARD W. HOOK, JUNIOR, SAMUEL O. POOLE AND W. F. FRIEDEWALD (*The Journal of Infectious Diseases*, May-June, 1949) have recorded virus isolation and serological studies on patients with clinical mumps during an epidemic in Atlanta in 1947-1948. They state that washings, cerebro-spinal fluid and hydrocele fluid were obtained, treated with streptomycin and penicillin, and inoculated into the amniotic cavity of fertile eggs, and these were incubated for five to six days at 37° C. Tests with agglutination of red cells were performed for the presence of virus, and inhibition of agglutination and complement fixation tests with known antisera for identification and for antibody titre in patients' sera were carried out. Of 17 samples of mouth washings, 10 yielded virus, and these were collected from patients within three days of the onset of parotitis. Seven samples of cerebro-spinal fluid, all of which had increased cell content, failed to show the presence of virus. Examination of one sample from a patient with severe orchitis showed virus to be present in testicular tissue, but not in hydrocele fluid. One mother whose baby was born during the acute stage of the disease had high antibody content in her serum, but the baby had neither virus nor antibody. The patient's serum titre early in the illness was usually less than 1:32, and the sample taken during convalescence was up to 1:1000, the commonest titre being 1:256; these results were similar both for hæmagglutination inhibition for complement fixation, and when applied to three different virus antigen preparations, a fact indicating that no differences could be demonstrated between three strains of mumps virus.

Lewis Blood Groups.

R. R. RACE, RUTH SANGER, SYLVIA D. LAWLER AND DOREEN BERTINSHAW (*The British Journal of Experimental Pathology*, February, 1949) have studied the Lewis blood groups of 79 families. They state that this antibody, discovered by Mourant in 1946, agglutinated 24 of 96 group O red cells, and thus appeared to be a new one. When tests were made of 571 unrelated persons, 22·7% were found to have the Lewis agglutinogen, and this appeared to have no relationship to the distribution of the A B O grouping. The results of the study of 79 families were classified; these on analysis supported the claim that adults whose red cells are Lewis-positive are genetically homozygous. The saliva of those with Lewis-positive red cells is usually of the non-secretory type, while in the owners of Lewis-negative red cells it is likely to be of the secretory type.

Penicillin in Human Milk.

R. ROZANSKY AND A. BRZEZINSKY (*The Journal of Laboratory and Clinical Medicine*, April, 1949) studied the excretion of penicillin in human milk in thirteen women two to eight days after delivery. A single large dose of crystalline penicillin G was given intramuscularly, and milk samples were taken before the injection and one, two, four and six hours later. In some instances, blood levels of penicillin were also estimated. After two hours a level of 0·2 unit per millilitre was found, and after six hours 0·3 unit per millilitre; the level gradually fell over a period of nine hours. In one patient, who had repeated doses, the level remained higher for a longer period, and one patient failed to excrete penicillin in the milk at all. The authors discuss the application of this piece of work to the treatment of mastitis.

Effect of Dyes on Influenza Virus A.

L. HOYLE (*The British Journal of Experimental Pathology*, April, 1949) has continued his studies of influenza virus by measuring the effect of triphenylmethane dyes on the intracellular growth of influenza virus A. The complement-fixation test was used to estimate the amount of soluble antigen in the allantoic fluid of eggs inoculated six hours previously with virus. Then groups of eggs were inoculated with virus followed thirty minutes later by quantities of dyes such as are used for intravital staining. It was found first that a group of acid dyes, such as brilliant cresyl blue and neutral red, had no effect on production of soluble antigen. The basic dyes, however, were more active, and could kill the embryo. Smaller doses led to diminution of production of soluble antigen, and retarded the rate of liberation of infective virus into the allantoic fluid, though it did not lower to any great degree the final titre. The dyes themselves first stained the allantoic membrane, but after a period of twenty-four hours the colour had disappeared. It would seem that these substances modify the intracellular metabolism of the virus particle in the phase during which it produces soluble antigen, possibly with the ribonucleic acid cycle.

Chloramphenicol in the Chemoprophylaxis of Scrub Typhus.

C. B. PHILIP, R. TRAUB AND J. E. SMADEL (*The American Journal of Hygiene*, July, 1949) state that they began a study of chloramphenicol (chloromycetin) in the chemoprophylaxis of scrub typhus with epidemiological observations on hyperendemic areas of the disease in Malaya. Three areas in the region of Kuala Lumpur were studied; the vegetation on two consisted of secondary jungle growth developing in fields formerly used for crops, the third area had been neglected for a longer period. Consideration of mite infestation of captured wild rats and exposed laboratory white rats showed that in some areas the number of vector mites was high, and they were present on all rats captured; in other areas the predominant species was not a vector of Rickettsia, and the greatest number of mites was collected in the morning hours between 8 and 11 a.m. The presence of rickettsiæ in the mites was shown by the inoculation of mice and the demonstration of typical lesions of the disease. This revealed a high rate of infection in the mites present with a strain of Rickettsia highly pathogenic to mice. Two attempts to isolate the organism from birds (quail) were also successful. In June and July, the number of mites recovered from rats was much smaller than during the rest of the experiment. Forty-six male volunteers were then assembled and exposed to infection in various parts of these three areas. Several hours on up to nine consecutive days were spent in the fields; again the wandering mites were most active early in the day. A few mites became attached to the skin of almost all the volunteers, and caused brief irritation and redness. Of the group, 29 contracted the disease. A second group of 29 were exposed to infection during July, and amongst these eight subsequently became ill with the disease.

Glomerulonephritis in Rabbits.

R. H. MORE AND D. WAUGH (*The Journal of Experimental Medicine*, May, 1949) have studied diffuse glomerulonephritis produced in rabbits by massive injections of bovine serum γ globulin. The animals were subjected to nephrectomy and rested for six weeks, and then given intravenously two doses of one gramme per kilogram of bovine serum globulin as a 10% solution in saline. Desensitization was carried out before the second injection. The control animals were allowed to survive nephrectomy for seven months before their tissues were examined

histologically. Of 18 treated animals, 14 had varying degrees of glomerulonephritis, microscopic examination of the morbid kidney showing reduction in the number of patent glomerular loops, some polymorphonuclear cells in the tufts, and acidophilic material in the tubules. In more severe lesions thickening of the capsules was seen, the most severe changes resulting in loss of many glomeruli, and in others crescent formation. An attempt was made to demonstrate antibodies to rabbit kidney in the serum of the treated animals, and serological evidence was obtained in two cases that these did exist. All animals had both serum antibodies and skin reactivity to γ globulin; but there was no correlation between these findings and the degree of nephritis present. Coagulation times were estimated in all cases, and it was found that after the injection of globulin there was a statistically significant increase in blood coagulability. The authors discuss the similarity of their results to those produced by other methods, and the inferences to be drawn in relation to the mechanism of the condition.

HYGIENE.

Manganese Pneumonitis.

T. A. LLOYD DAVIES AND H. E. HARDING (*British Journal of Industrial Medicine*, April, 1949) give a short résumé of a reported case of manganese poisoning. Observations by the first author have shown a causal relationship of manganese to the high incidence of pulmonary disease in a group of men, numbering from 47 to 124 and observed over a period of eight years, exposed to the inhalation of manganese oxides. There was a high incidence of pharyngitis, bronchitis, bronchopneumonia and an illness similar to pneumonia, for which the manganese pneumonitis was suggested. The particle size of the manganese oxide dust was very fine. Approximately 80% of the particles were less than 0·2μ in diameter. Because of their fineness the particles were particularly liable to give rise to pulmonary effects, but sufficient manganese was not absorbed to cause systemic poisoning. Animal experiments carried out by the authors showed that the intratracheal injection of suspensions of manganese dioxide in normal saline and of solutions of manganese chloride in normal saline into the lungs of rats causes characteristic histological changes. Within fifteen minutes of contact with manganese dioxide the epithelial cells of the bronchi discharge their mucus, and the epithelium becomes ragged and may be loosened from the basal membrane; an intense mononuclear-cell infiltration of the alveolar walls and alveoli develops after about twenty-four hours, and shortly after this large hydropic cells appear, often in large numbers; late and inconstant changes are a granulomatous reaction and giant-cell formation; after about a year these changes have disappeared and the lung appears normal. The injection of manganese chloride causes intense congestion of the lung; many animals die from pulmonary œdema; the bronchial epithelium is disorganized and often detached, but compared with the effect of manganese dioxide the histiocytic (mononuclear) infiltration of the alveoli is less intense. In man and animals, manganese dust in suitable particle size introduced into the respiratory system will, without the presence of other factors, cause pneumonitis.

Nutrient Retention during Canned Food Production.

E. CAMERON, R. PILCHER AND L. CLIFCORN (*American Journal of Public Health and The Nation's Health*, June, 1949) describe how operations commonly involved in the home preparation or in the commercial processing or preservation of food may affect the initial nutrient compositions. They state that a considerable volume of literature has been developed on nutrient retention during commercial canning, and the data obtained in major studies in this field are summarized and discussed. Tables are given showing the ascorbic acid, carotene, niacin, riboflavin and thiamine content of canned foods. The effect of the blanching process on these substances is also shown in table form. The authors state that vitamin retentions vary with the product and nutrient under consideration. However, raw products with a significantly high content of specific nutrients remain significant sources after canning. Certain operations, notably blanching, are subject to improvement, and studies of this type currently in progress are described. The newer types of high-short thermal processing show considerable promise of improved retention of heat-labile vitamins in products to which such processes may be applied.

The Nutritive Values of Processed Grain Foods.

F. NORDSIEK (*American Journal of Public Health and The Nation's Health*, June, 1949) discusses milling, manufacture, destruction and restoration of nutrients during these processes, and supplementing of nutrients to augmented levels in the processing of grain foods. He states that, in general, consumers demand grain foods in milled and manufactured form. These processing operations reduce certain nutrients present in the original grains. Since cereals are a major food, this nutrient loss originally constituted a serious nutritional problem. The practical answer to this problem has proved to be enrichment, restoration, or fortification, mainly by the use of synthetic vitamins and assimilable mineral salts. The rationale followed in establishing enrichment levels has led on occasion to concentrations of nutrients higher than those in the natural whole grain. This fact, *plus* the customary use of supplementary nutritive ingredients in the making of certain grain foods, means that in some respects the milled and manufactured products may be more nutritious than the grains from which they are made. Tables are given showing the thiamine, riboflavin, niacin, iron and calcium content of whole wheat bread, white bread and enriched white bread.

Talc Pneumonokoniosis.

A. I. G. MCLAUGHLIN, ENID ROGERS AND K. C. DUNHAM (*British Journal of Industrial Medicine*, July, 1949) describe pneumonokoniosis due to talc in a man who operated a tire-extruding machine for about thirty years in a rubber tire factory, where he was exposed to a fair concentration of talc dust; the cause of death was (i) heart failure due to rheumatic endocarditis and (ii) pneumonokoniosis. A description is given of the macroscopic and microscopic lung appearance and of the mineralogy of the lung contents and the talc used in the factory. Moderately advanced pneumonokoniosis of both lungs was found at autopsy. This condition had been diagnosed during life and demonstrated radiographically. A feature of the histological appearances were "curious bodies" similar to asbestosis bodies associated with the pneumonokoniotic lesion. Though there was much fibrosis of the lungs and abundant doubly refractile particles, an X-ray analysis of the lung ash suggested that there was less than 0·06% of free silica in the lung (dry weight). The talc in the lungs consisted almost entirely of straight fibres, with a few curved fibres and shreds. This suggests that the respiratory passages acted as a selective filter, allowing the fibres to get into the lungs while rejecting the plates. The mineralogical observations establish clearly that fine grinding of talc gives rise to material having individual fibre form, and perhaps suggests the presence of a previously unsuspected second cleavage parallel with the Z-direction. It is likely that talc pneumonokoniosis and asbestosis are similar diseases, though asbestos appears to be more actively fibrogenic than talc. The evidence suggests that talc pneumonokoniosis is caused only by the fibrous varieties of talc.

Acute Pneumonitis Associated with Beryllium.

JOSEPH C. AUB AND ROBERT S. GRIER (*The Journal of Industrial Hygiene and Toxicology*, May, 1949) report seven cases of acute pneumonitis in workers exposed to dusts and fumes of pure beryllium metal and beryllium oxide in a metallurgical laboratory, where they were engaged in melting, casting, machining, grinding and welding very pure metallic beryllium or making castings and moulds with finely divided beryllium oxide. In six cases, symptoms appeared in eight to ten weeks. Shortness of breath "on one flight of stairs" to severe dyspnœa on any exertion, excessive fatigue and lassitude were the most frequent and striking symptoms. Dull chest pain, dry cough, marked loss of weight, gastro-intestinal symptoms and fever occurred. Physical and laboratory findings were indefinite. The duration of X-ray findings was five to six months, and varied from increased perihilar shadows to extensive granular infiltration suggestive of acute silicosis, or sarcoidosis similar to the chronic pulmonary granulomatosis associated with fluorescent beryllium phosphors. The progression from increased hilar markings through ground glass appearance to extensive infiltration of a granular nature, and then back to normal, was seen in several cases. It is pointed out that these cases are interesting because they represent the first examples of this reaction occurring after exposure to only the oxide and the metal, in contrast to other reported cases in which the soluble salts of beryllium were also present. These cases tend to support the view that the beryllium ion is toxic in its own right, rather than attributing the disease associated with soluble beryllium salts to the acid radicals.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on September 22, 1949, at Sydney Hospital. The meeting took the form of a series of clinical demonstrations by members of the honorary medical and surgical staffs of the hospital. Part of the account of this meeting appeared in the issue of November 5, 1949.

Bronchiectasis.

DR. W. L. CALOV showed a female patient, aged eighteen years, a stenographer by occupation, who had reported on April 5, 1949, with the history of cough and sputum-production for seventeen years. She had had pertussis as a baby with cough ever since and severe colds every winter. She had had Bright's disease when aged eleven years and pneumonia when aged ten years. Her tonsils were removed at the age of eleven years. At the time of the meeting she was coughing up four ounces of sputum daily; the sputum had been streaked with blood on and off for two years. Examination revealed that she was an apparently healthy girl, but definite clubbing of the fingers was present. She had bronchitic signs at both lung bases, and although an X-ray examination of the sinuses showed some dulness, an ear, nose and throat consultant could find no active sepsis. Bronchography revealed well established digitate bronchiectasis on the left side involving all the lower main bronchi and the linguia. The middle right basal bronchus was also affected. She was unable to manage instillation of penicillin and was being treated with breathing exercises, postural drainage and sulphadiazine, one gramme daily with hæmatological control.

Dr. Calov then presented a female patient, aged eighteen years, who first sought advice in January, 1945, when she was aged fourteen years. At that time she complained of cough and sputum-production ever since she was a small child. Occasionally her sputum was tinged with blood. Her past history revealed that she had had measles and rheumatic fever, but no pertussis. Her tonsils had been removed when she was six years of age. She had been referred from another hospital with the report that she had "saccular" bronchiectasis at the base of the left lung. Examination then revealed a healthy looking girl with no clubbing of the fingers. Her weight was six stone three pounds and her height five feet three inches. There were post-tussive crepitations at the base of the left lung. After several visits she failed to report again until May, 1949. She then stated that she had been fairly well, but had been coughing up half a cup of sputum daily. Examination revealed a healthy looking girl. Her weight was seven stone five pounds and definite clubbing was present. There were post-tussive râles at the base of the left lung, and ear, nose and throat consultation revealed chronic infection of the maxillary sinuses. A bronchogram on July 4, 1949, revealed well established saccular bronchiectasis involving all the lower left main bronchus. The lingula and the right lung were not affected. At the time of the meeting she was having weekly bronchoscopic aspirations and ear, nose and throat treatment. She was later to undergo lobectomy on the left side.

Pulmonary Tuberculosis.

Dr. Calov's next patient was a male, aged thirty years, who had been diagnosed as suffering from pulmonary tuberculosis in 1944. He was admitted to a sanatorium, where he was treated by artificial pneumothorax of the left side. That was suspended, and phrenic crush was performed. He was first examined by Dr. Calov in June, 1947. His general condition was then good; but X-ray examination showed a large cavity in the mid-zone of the left lung and collapse of part of the upper lobe of the right lung. Tubercle bacilli were found in the sputum. He was advised to have thoracoplasty. In the period from November, 1948, to January, 1949, three stages of thoracoplasty were performed. He was readmitted to the sanatorium, but stayed one day only. At the time of the meeting he felt well. He had very little sputum, his blood sedimentation rate was four millimetres in one hour, his weight had fallen a little, but his general condition was good. Dr. Calov considered that the result might be regarded as satisfactory.

Dr. Calov then presented a male patient, aged forty-eight years, who had been diagnosed as having pulmonary tuberculosis in 1932. His first symptoms had appeared in 1929, when he had hæmoptysis. Thoracoplasty on the left side was performed in 1937. In the same year cold abscesses appeared and ruptured from the lower, anterior part of the right hemithorax. An ischio-rectal abscess was opened in 1948. He had been examined in the urological department for frequency of micturition. X-ray examination showed irregularity of the calyces of the upper pole of the left kidney. Tubercle bacilli were recovered from the pelvis of each kidney. His general condition was fairly good despite widespread tuberculosis. His left hemithorax moved very little on respiration and practically no breath sounds were audible on the left side. Depressed adherent scars were present on the anterior aspect of the lower part of the right hemithorax. The epididymides were nodular and tender. A skiagram of the thorax showed complete dulness of the left side and some scattered calcified lesions of the right lung field. The blood sedimentation rate was 14 millimetres in one hour.

Medical Societies.

THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING of the Medical Sciences Club of South Australia was held in the Anatomy Lecture Theatre, Frome Road, Adelaide, on October 7, 1949.

"Q" Fever in South Australia.

DR. J. M. DWYER and MISS J. STOKES discussed "Q" fever with particular reference to its occurrence in South Australia. It was stated that "Q" fever had not been recognized in South Australia before December, 1947, but at that time several cases of an acute febrile disease came under the notice of Dr. Dwyer. The patients were all abattoir workers, and they had typical symptoms of "Q" fever. In February, 1948, five patients, all abattoir workers with similar symptoms, were admitted to the Royal Adelaide Hospital. Sera from those patients and also from Dr. Dwyer's patients were sent to the Queensland Institute of Microbiology, and were shown to have developed agglutinating antibodies to "Q" fever.

Two strains of "Q" fever had been isolated from the blood of febrile hospital patients. Defibrinated blood was inoculated into mice intraperitoneally, and post-mortem examinations performed ten to twelve days afterwards showed slight enlargement of their spleens, but rickettsiæ were not seen in smears. Ground-up spleens were inoculated into the yolk sac of seven-day developing chick embryos, and a very light infection occurred. The rickettsiæ were well adapted to the yolk sac after several passages, and from this material ether extracted yolk-sac antigens were prepared, after the methods of Craigie, and Topping and Shepard. Guinea-pigs inoculated with patients' blood developed complement-fixing antibody to "Q" fever after a mild febrile episode.

A survey for immunity to "Q" fever had been performed on the sera of 80 volunteers out of 120 of the abattoir workers of the beef section by means of complement-fixation tests. Amongst these 28 "positive" sera were detected, giving an incidence of 35%. Three laboratory infections had been investigated, and besides those one subclinical infection had been detected amongst 35 staff members.

It was pointed out that "Q" fever had been first described in Queensland by Derrick in 1937, and since then had been described in the United States, Panama, Central and Southern Europe and North Africa, with a few isolated cases in England. The strains isolated in those areas were antigenically similar to Australian strains, but tended to produce a rather more severe disease in man, with which an "atypical" pneumonia was often associated, beside the classical symptoms of the Australian disease—acute headache, rigors, stiffness of the neck, aching joints and nausea.

Vagal Influence and the Respiratory Centre.

MR. D. I. B. KERR discussed vagal influence and the respiratory centre. He stated that two problems in respiratory physiology were (i) whether the respiratory centre was inherently rhythmic, and (ii) how the vagal influences modified the activity of that centre. There were three experimental approaches to the problem of inherent rhythmicity in the respiratory centre: ablation of the brain stem, stimulation of tracts and nuclei, and chemical stimulation of the central nervous system. From ablation experiments it was considered that the medulla contained a centre capable of unrestrained inspiratory activity in the absence of an upper pontine inhibitory mechanism and/or

vagal inhibitory influences arising from inspiratory distension of the lungs. Confirmatory stimulation experiments located inspiratory and expiratory areas within the medulla. However, stimulation failed to exhibit a specific respiratory inhibitory mechanism in the pons. It became apparent that the brain stem contained a system extending from the basal ganglia to the cord which was capable of either facilitating or inhibiting somatic and autonomic activity. Respiratory responses due to stimulation of the reticular formation must, as Bach had shown, be considered in the light of those generalized activities and due caution be taken before definite respiratory areas were assigned in the brain stem, since the respiratory motor neurons were influenced along with the general somatic outflow.

The reticular formation might well be responsible for the phenomenon of apneusis, there being some affinities of apneusis and decerebrate rigidity as shown by ablation experiments, as well as by stimulation, with cyanide or carbon dioxide, to which the facilitatory mechanism was sensitive.

Hoff had shown that apneusis was superimposed on rhythmic respiration and was not an invariable consequence of low pontine transection with vagal block. Wyss had located medullary areas which mediated the inspiratory excitor effects of low frequency central vagal stimulation and the inhibitor action of similar high frequency stimulations. Both those effects were influenced by a system which extended into the upper part of the pons as was shown by Trevan and later by Meier and Bucher. Gordh had been able to confirm those facts, finding also that deep ether anæsthesia caused a "central" vagotomy with an ascending paralysis of the respiratory musculature. In Adelaide they had been able to confirm that observation and find that mere section of the vagi during light anæsthesia could produce essentially similar results, characterized by a failure of coordinated activity of thorax and diaphragm. Those results would be described in greater detail elsewhere.

It was concluded that the vagal lung afferents influenced the respiratory pattern by a system which extended into the mid-brain, was responsible for coordination of individual respiratory movements and depended upon afferent frequency for its effects.

Zinc in Plants.

PROFESSOR J. G. WOOD described experiments carried out by Miss P. M. Sibly and himself in which oat plants were grown in Laffer sand or in water culture containing varying amounts of added zinc. In each case the zinc contents of living and dead leaves, stems, roots, inflorescence and grain were determined at intervals during the life cycle of the plant.

He stated that zinc was absorbed continuously throughout the life cycle. It was shown that there was a threshold value of zinc for grain production in oats, but large increases in zinc content of the plant did not cause proportionate increase in either dry matter or grain at final harvest.

Leaves contained 20% to 30% of the total zinc in the plant, but that zinc was not translocated from the leaf to other organs during senescence; nor could zinc be removed by dialysis of macerated leaf material. Of the total amount of zinc in the leaves 15% to 20% was localized in the chloroplasts in oats, but in spinach 50% of the total leaf zinc occurred in the chloroplasts.

The amount of zinc in roots increased up to the time of appearance of the inflorescence, but thereafter decreased and was translocated from the root. The zinc content of the inflorescence and grain was accounted for by translocation from the root and uptake from the medium. Relatively large amounts of zinc might be stored by the roots. Carbonic anhydrase activity of leaves had been determined at various times during the life cycle and the relation of that activity to zinc content was hyperbolic in form.

Correspondence.

CARCINOMA OF THE THYREOID GLAND WITH REPORT OF TWO CASES.

SIR: It appears that in neither of Dr. Hoddle's interesting cases of carcinoma of the thyreoid (THE MEDICAL JOURNAL OF AUSTRALIA, October 15, 1949) was the diagnosis made before operation, though in both metastasis had occurred, in the one case to the lungs and in the other to the cervical lymph nodes. The diagnosis is in fact often missed because, in relation to other forms of goitre, thyreoid cancer is rare and, as in Dr. Hoddle's cases, the gland may not proclaim its malignant character.

The outstanding recent contribution to this subject is the work of King and Pemberton (1942). They demonstrated that "lateral aberrant thyreoids" are in fact lymph-glandular metastases from a primary cancer in the homolateral lobe. Dunhill and earlier workers were misled into believing them to be "lateral aberrant thyreoids" become malignant, by the apparent absence of disease in the thyreoid, but King and Pemberton showed that the homolateral lobe may not be enlarged and may even require to be sectioned if the primary growth is to be demonstrated. It was thus established that at biopsy of cryptogenic malignant glands (such as existed in Dr. Hoddle's second case) the corresponding lobe of the thyreoid should always be explored and, if suspicion persists, removed.

The only positive diagnostic criteria of thyreoid cancer are biological, especially evidence of growth into surrounding structures (including a major vein) and lymphatic or hæmatogenous metastasis. Reports from certain United States clinics, quoted by Dr. Hoddle, that about 25% of single thyreoid nodules are cancers, almost certainly depend on dubious microscopic criteria, including the finding under high magnifications of isolated cells in what might be a venule or of a small area of supposed capsular invasion. The point is that so high a figure is at complete variance with the known low incidence of thyreoid cancer, though hosts of people must carry single nodules about, many even being blissfully unconscious of their presence. However, even if, as seems likely, the incidence of malignancy in such nodules is nearer 1% to 2%, there is still every justification for recommending their routine removal, as in the best hands the risk is quite negligible.

Yours, etc.,

135 Macquarie Street, Sydney,
October 31, 1949.

FRANCIS F. RUNDLE.

Reference.

King, W. L. M., and Pemberton, J. de J. (1942), "So-called Lateral Aberrant Thyroid Tumours", *Surgery, Gynecology, and Obstetrics*, Volume LXXIV, page 991.

POSTURE FUNDAMENTALS.

SIR: Dr. Jean Macnamara's article entitled "Posture Fundamentals" in THE MEDICAL JOURNAL OF AUSTRALIA of October 22, 1949, is full of interest and should be most useful to those concerned with the care of infants and children.

The reason, however, for this letter is Dr. Macnamara's statement that posture is "a subject which merits more attention than it receives in the medical curriculum . . ." My particular interest in her article centres round the words "to act, advise and co-operate in the prevention of crippling diseases" in the constitution of the Australian Orthopædic Association, to which she referred in an earlier article.

Although there is any amount of evidence as to the importance of posture, one finds that there is very little unanimity regarding its nature. Thus, Professor Raymond A. Dart, writing in the *South African Medical Journal* of February 8, 1947 ("The Attainment of Poise"), states: "To elaborate a logical therapeusis for malposture, its real nature must be apprehended. We must agree first of all that, in using the word 'poise' we know what is under discussion. We should also be able to elaborate the essential terminology without misusing, by tautology or qualification that is misleading, words so full of meaning as 'posture' and 'poise'!" Professor Dart in his article quotes statements by various orthopædists which clearly reveal the confusion brought about by the varying ideas on poise and posture.

Professor Dart also quotes Sir Charles Sherrington as saying: "Mr. Alexander has done a service to the subject by insistently treating each act as involving the whole integrated individual, the whole psychophysical man. To make a step is an affair, not of this or that limb solely, but of the total neuromuscular activity of the moment—not least of the head and neck."

Much information is available on posture, but a very great deal of it concerns posture in various animals, and opinions on posture in man vary. Furthermore, from the literature in Great Britain, one receives the impression that there is diversity of opinion as held by pædiatricians and orthopædists on the true nature of posture. It is therefore obvious that an essential requirement for the teaching of posture in the medical curriculum in the various age

groups is a suitable text-book setting out clearly the views of the majority of those qualified to speak, and the reasons for dissent from those views by the minority. Dr. Macnamara has stated that it is the function of the Australian Orthopædic Association "to act, advise, and co-operate in the prevention of crippling diseases". Here is a task for them that has long been awaiting attention.

I do not wish it to be thought that in the University of Queensland no attention is given to the teaching of posture. Numerous aspects of the problem are dealt with, notably in the departments of surgery, obstetrics and social and tropical medicine. In addition, medical students in their first year, study and participate in physical education under the direction of the director of the department of physical education.

Be this as it may, I do not think it can be too strongly emphasized that a clarification of the subject is badly needed, together with a small text-book in which are set out the principles of the subject, particularly as they concern the general practitioner in his everyday work.

Yours, etc.,

E. S. MEYERS,
Acting Honorary Professor,
Social and Tropical Medicine.
The University of Queensland Medical School,
Herston Road,
Brisbane, N.1.
October 24, 1949.

MEDICAL FEES.

SIR: I would ask my medical confreres to ponder these facts: In 1938 the Sydney basic wage was 81s.; in 1949 130s. In 1938 the six capital cities' average was 78s.; today 127s. In 1938 the medical fee was 10s. 6d.; in 1949 it is 10s. 6d. The "invisible labour" of a medical practice—the doctor's wife—is paid today what she was in 1938—nothing. Costs—wages, drugs, car, petrol *et cetera*—have skyrocketed, and, with the devaluation of the pound, will go still higher.

Is it not high time the scale of medical fees be revised? One suggestion is the fee be adjusted proportionate to the basic wage increase, and be pegged to it, to fall or rise to the nearest sixpence.

In these days of proposed national health schemes it is to be hoped this question of fee adjustment is being given due consideration. Or is it?

Yours, etc.,

"MEDICUS".

SOME PROBLEMS ASSOCIATED WITH THE MANAGEMENT OF CARCINOMA OF THE BREAST.

SIR: Dr. Nelson, in his recent letter upon the treatment of carcinoma of the breast, has reminded us of the effective palliation which radiotherapy may give to the patient with metastases. Some years ago, one of my patients developed a metastasis, in front of a sterno-clavicular joint, and the hard fixed lump disappeared under radiotherapy. This impressed me. But at about the same time I was given the care of a patient at Saint Vincent's Hospital who had had a radical mastectomy performed elsewhere, and who now had multiple secondary growths in the skin flaps. Most of the growths were in the area discoloured by prophylactic radiation, and these did not appear to be any smaller or less vigorous than those appearing in the skin of normal colour. This puzzled me. But the probable explanation appeared when I discovered that the optimum dose given to each unit volume of tissue of a well-localized metastasis is very much greater than the dose given to each unit of tissue in prophylactic pre- or post-operative radiotherapy. It is impossible to give a desirable dose to each unit of tissue in the chest wall, internal mammary glands, skin flaps, axilla, retro- and supraclavicular region *et cetera*. This suggested to me that prophylactic radiotherapy is an irrational procedure. At a recent meeting I asked the radiotherapists for a statement regarding the comparative doses delivered to each unit volume of tissue in radiotherapy delivered (i) pre-operatively to a point, for example, in the capsule of the sterno-clavicular joint, which has no clinical evidence of carcinoma, (ii) post-operatively to the same point in the same state, (iii) to the same point when affected by a visible palpable metastasis. But, instead of the hoped-for answer in physical units, I was told that a good radiologist will even risk necrosis and the skin flaps may be very thin. My question was thus evaded.

We must avoid the tendency to assume a value for prophylactic radiotherapy based upon the results of treatment of a well-localized metastasis.

Dr. Nelson, like other radiotherapists, stresses the need for precision and scientific accuracy in radiotherapy. But the radiotherapist, in prophylactic work, is like the mathematician who would fix a straight line when he is given only one point. He can fix the tube end of the line. But the other end is a cancer cell which may not exist, but which, if existent, is of unknown whereabouts, of doubtful radioresistance, but probably resistant, and imbedded in tissues of unknown tolerance. Moreover, even if the radiotherapist knows the exact location of the cancer cell, how can he precisely apply the optimum cancericidal dose, when radiotherapists differ as to what is the optimum cancericidal dose? If then accuracy is essential to this work, and at the same time unattainable, we have a second reason for believing that prophylactic radiotherapy in breast carcinoma is irrational.

This method of reasoning is deductive. In this matter it is the best method available, for the other method, the inductive, has for its material inconclusive and confusing statistical data.

Ralston Paterson, addressing the meeting of a hospital staff during his visit to Sydney, told us that we were ten years behind the intensive activity in England, in regard to new methods of treatment. The issue of the *British Medical Journal* current at that time reported the comment of the Ministry of Health upon the British vital statistics, that the cancer mortality remained the same in spite of the activity of recent years. The latest statistics reveal an increase in the cancer mortality. One wonders if this is due to a diversion of patients from the old well-tried method of block removal to newer less effective methods.

Radiotherapists have "demonstrated" many things, for example, that pre-operative radiotherapy should be given up. Richards, however, in *The British Journal of Radiology* (1948) presents 1271 cases, divides them up according to a clinical index of malignancy and "demonstrates" a 25% to 30% gain for pre-operative therapy in two groups of cases. But closer study shows that in these two groups there are but 15 and 52 patients. In another group with 100% cures there were three patients.

In the *Annals of Surgery* of June, 1947, two series of similar numbers of radical mastectomies are reported, one by Finney, of Baltimore, with irradiation in only very few cases, and one by Marshall, of the Lahey Clinic, with routine irradiation. There was practically no difference in results.

During 1942 and 1943, Haagensen and Stout, from the Presbyterian Hospital in New York, studied 1040 cases and concluded that pre-operative or post-operative irradiation did not improve results.

McWhirter in Edinburgh, quoted by Dr. Nelson, has "demonstrated" that the radiation of axillary glands is better than their dissection. But Adair, in charge of the breast clinic at the Memorial Hospital in New York, analysing 13,000 cases, found that irradiation of the axilla is quite unreliable, and may cause death of the cancer cells in one gland and leave those in a neighbouring gland quite viable. In his clinic they dare not deliver a reliable cancericidal dose to the axilla, for fear of lymphœdema, and they find that this complication becomes progressively worse and that little relief is available for the sufferer (*Annals of the Royal College of Surgeons*, June, 1949). On the other hand, the lymphœdema following operation without radiotherapy tends to get better and is rarely crippling.

In the years from 1935 onwards Stuart Harrington, of the Mayo Clinic, followed up with remarkable thoroughness 3381 patients. He stated that "roentgen therapy has been of no significant aid as an adjunct to radical surgical treatment and should be used not as a routine treatment, but only if the malignancy is of high grade. The end-results in cases in which the patients have died show that the patients who were subjected to surgery only lived longer than those who were subjected to both surgery and roentgen therapy. In these cases it is evident that roentgen therapy was not an aid to surgical treatment and may have been detrimental to it in some cases". (The growths for which he advocated radiotherapy were histologically anaplastic—a very small group.)

There is another point which needs clarification. Radiotherapists, like Dr. Nelson, hint rather mysteriously that there have been modern improvements in radiotherapy. The suggestion is sometimes made, sometimes implied, that even if the results fifteen years ago were bad, they should be better today. But it may fairly be asked, has there been

any fundamental improvement in radiotherapy over the last fifteen years? What makes the X rays of today more lethal to the cancer cell than they were fifteen years ago? Surely it is not a shorter wave-length? Radium delivers a ray with shorter wave-length, and has been tried and abandoned in breast cancer.

The present evidence suggests that routine prophylactic pre- or post-operative radiotherapy in carcinoma of the breast is an unjustifiable gesture, nearly always futile, sometimes harmful and rarely useful.

Yours, etc.,
V. J. KINSELLA.

235 Macquarie Street,
Sydney,
October 31, 1949.

A REVIEW OF FIFTY CASES OF GASTRECTOMY FOR PEPTIC ULCER, WITH SPECIAL REFERENCE TO LOCAL ANÆSTHESIA.

SIR: I was very interested to read Mr. V. J. Kinsella's review of fifty cases of gastrectomy for peptic ulcer, with special reference to local anæsthesia (October 22, page 595).

In my opinion the article merely supports the views long held by those interested in regional analgesia, that previous to the advent of modern anæsthetic methods and properly trained anæsthetists, regional methods of anæsthesia gave better results than the methods of general anæsthesia then available. The points brought out in the final paragraph of his article are to be found in any of the text-books on regional analgesia published before the war. The position is now, of course, radically altered, especially since the advent of the relaxing agents, which if carefully used have revolutionized general anæsthesia. Today prolonged intraabdominal operations can be performed on poor risk patients or even in cases of acute hæmorrhage with better results than the former superior regional methods. Having once written a small text-book extolling the virtues of regional analgesia, I cannot now be accused of bias.

It is a pity that Mr. Kinsella has not had the services of a competent anæsthetist trained in modern methods, because, if he had, he would not be making claims which were only valid several years ago. It is only fair that the reference to the Royal Melbourne Hospital should have stated the year in which these figures were published, because as the review stands it is obsolete, inaccurate and grossly misleading.

Yours, etc.,
NORMAN R. JAMES,
Director of Anæsthesia.

The Royal Melbourne Hospital,
Melbourne,
October 26, 1949.

A CANCER INSTITUTE IN VICTORIA.

SIR: The Parliament of the State of Victoria on December 13, 1948, passed an Act to establish a Cancer Institute. It will serve the needs of the States of Victoria and Tasmania which contain, approximately, a population of two million persons. The Institute will be established primarily as a radiotherapeutic centre.

I quote for your information the preamble to the Act:

> An Act relating to the establishment and management of a Cancer Institute for the purposes of research into and treatment of cancer and allied conditions, and for other purposes.

The establishment of a Central Institute for the treatment of cancer by radiotherapy has been in the forefront of the activities of the Anti-Cancer Council of Victoria for the past twelve years.

The passing of the Act of Parliament without amendment in both Houses was a demonstration of the acceptance by the Members of Parliament of the propositions which had been advocated by the Council. The formation of the Institute is an attempt to centralize the radiotherapeutic facilities and clinics of the major hospitals of the two States in a Central Institute in order to achieve higher efficiency and more economical use of available apparatus. The practising radiotherapists of Victoria are also being offered facilities to establish their private practices within the Institute.

The Government of Victoria has been enabled to make available for the treatment purposes of the Central Institute a fine site in Melbourne with modern hospital buildings which had been vacated by the transfer of one of the smaller public hospitals to a larger site. These buildings comprise a modern private and intermediate block of 70 beds, a block of modern wards containing 75 beds for public patients, an out-patient department and a nurses' home. In addition to these modern buildings there is a central block of older buildings which house the administrative staff and sufficient space for temporary use for X-ray diagnostic purposes.

A special committee set up by the Minister of Health of Victoria examined these buildings and reported to the Government of the State that in the opinion of the members it was a practicable proposition to establish on this site a modern radiotherapeutic institute. Preliminary plans were drawn up by the Government Architect for a new out-patient department, for a radiotherapeutic department containing eight X-ray therapy machines, a physics department, a mould room, records office, almoner's office *et cetera*. The committee recognized that many other alterations would be necessary, but it was evident from the plans that the present buildings could be suitably adapted.

In March of this year the Government of Victoria appointed a Cancer Institute Board which would have the responsibilities of determining policy and an executive committee consisting of four members of the board and three executive officers was established for administrative purposes.

It is hoped that appointments to the positions of medical director and assistant medical director will be announced at an early date.

A building subcommittee has been established by the board to consult with the chief Government Architect when the plans for the major reconstruction work have reached a point where expert advice is necessary.

It is unlikely, owing to the shortages of building supplies and of labour, that the necessary work will be completed for from eighteen to twenty-four months.

A long-range plan is the demolition of the central block and the erection of a modern building with full provision for research. The whole question of research into the many problems connected with cancer has caused the board much concern. The decision has been made to obtain the services of a director of research of international standing and to build his department around him.

This provision for research covers the diagnostic, physical and therapeutic fields, and it is the expressed intention of the board to provide ample opportunities for scientific workers to contribute to the general advancement of the knowledge of cancer and consequently the attainment of its control. The institute will undertake the training of medical graduates in the field of radiotherapeutics, and when they have attained the required standard of efficiency is prepared to send them overseas for further experience in the specialty.

Under the provision of the Act of Parliament finance for the reconstruction of the premises and maintenance will be provided by the Government of the State of Victoria.

Yours, etc.,
C. V. MACKAY.

Cancer Institute,
483 Little Lonsdale Street,
Melbourne, C.1,
Victoria.
October 14, 1949.

SYPHILIS RESURGENS.

SIR: I read with keen attention the most interesting article on "Syphilis Resurgens" by E. H. Molesworth (this journal, October 1, 1949). It is just the paper I was looking for in the last couple of years, a paper which would awaken the somewhat slumbering interest in syphilis. I think the author can feel successful. It would, however, develop into an even greater, and in its consequences more far-reaching, success if it could open up a good chance for discussing syphilis. In my opinion every official quarter should now make a statement on this question of utmost significance, and everyone who feels he has something to contribute. It is in the interest of the public health of the community as much as of science. Cooper Booth (1949) was the first with his declaration, I wish to be the next one to follow in his steps. I would comment as a specialist of internal diseases.

I am afraid Dr. Molesworth was not quite correctly informed that "cases of cardiac involvements, aneurysms and other diseases of the arteries and the circulatory system have become very rare". I had similar information myself when I settled here two and a half years ago and began to query about syphilis in Australia. The aforenamed diseases are, in fact, not rare in Sydney, or in

Australia, at all, just as *tabes dorsalis*, general paralysis of the insane and other involvements of the central nervous system are not rare, though "sharply diminished in number". Congenital syphilis has decreased to a large extent, but did not "practically disappear". Interesting cases of syphilitic aortitis, of aneurysm or neurosyphilis are to be seen at every hospital meeting of the British Medical Association and frequently at staff meetings of the large hospitals.

Let us collect some official data on aortitis, the most significant manifestation of late syphilis, the condition *kat'exochen* which makes syphilis a dangerous disease incomparably more than any other condition provoked by it, including general paralysis. According to the report of the post-mortem room of the Sydney Hospital (for which I have to thank Dr. A. A. Palmer) 19 cases of aortitis and aneurysms have been found amongst the 1176 dissections performed between January 1, 1935, and January 1, 1940 —that is, 1·61%. The corresponding figure for the interval 1940-1947 is 1·63%. In the fourth thousand of a series of autopsies performed at the Adelaide Hospital (Carver, 1940), 20 cases of aortitis and aneurysms were found—that is, 2%. C. Duncan (to whom I am grateful for his personal communication) found 20 cases of syphilitic aortitis and aneurysms amongst 1880 autopsies performed in the last twelve years in Hobart—that is, 1·06%.

The official death statistics of the Commonwealth (Commonwealth Bureau of Census and Statistics, 1946) give the following figures for the years 1943-1946:

Syphilitic Condition.	Males.				Females.			
	1943	1944	1945	1946	1943	1944	1945	1946
Locomotor ataxia ..	30	22	19	19	3	2	4	5
General paralysis of the insane	64	69	42	40	22	14	12	9
Aneurysms	169	153	124	141	55	39	49	39
Congenital syphilis ..	6	6	9	5	9	5	4	2
Others	63	66	54	64	23	22	17	20
Total	332	316	248	269	112	82	86	75

The totals for both sexes are: 1943, 444; 1944, 398; 1945, 334; 1946, 344.

We find a slow decline in almost every column. I feel, however, that the totals 444, 398, 334, 344, representing the mortality of syphilis for the respective years, are far too low. The mortality rate of syphilis is, without doubt, higher; for the figures do not include deaths due to pure aortitis, which are hidden in the mass of the heart diseases and coronary diseases (10,729 and 4951, respectively, in 1943). Let us remember that aneurysms develop only in about 10% of aortitis cases: "Syphilis accounts for 10 to 12% of the total deaths from cardiovascular disease" (Stokes, Beerman and Ingraham, 1944). Keeping all this in mind, I estimate the number of deaths due to aortitis to at least two or three times higher than the figures given above, and it may amount probably to even more. Accordingly, the morbidity of cases of *aortitis syphilitica* amounts to several thousands in every year.

To my mind all these data—which are distinctly lower than the corresponding data in most other countries—are quite natural. They are due to the tide of new cases of syphilis described by Molesworth for the second and third decades of this century, as the late manifestations of syphilis appear fifteen to thirty years after the infection. I am afraid it will take some more years before the last remnants of the recessing wave of the described new cases of syphilis will disappear completely.

But all these data are to my mind very important from another point of view too. It is a sign that so far we have not succeeded at all to "eradicate entirely the infective agents". The combined cure with arsenical and bismuth drugs might have been indeed a very important progress in the therapy of syphilis, a significant milestone, but it is very far yet from being the final solution. There is certainly urgent need for a radical completion of the therapy of syphilis, and in regard to the new tide of syphilis of the last years in Australia, described by Molesworth and Booth, the need is of imperative urgency. Let us hope that penicillin will contribute its share. In some official quarters in the United States of America the hopeful promise has been made that syphilis might be eradicated completely by about 1970. I wish this to be true with my full heart; it would be a very big blessing for the human race.

But I have to confess that I feel distinctly sceptical both about the predicted time and about the completeness of eradication. I feel that to achieve this aim there would be need for a much more concentrated and energetic attack against the disease. The attack would have to be fought not only by the medical profession, aided by intensive cooperation of pharmaceutical research, but by education with the help of State and society, together, shoulder to shoulder, in a final effort. If this attack failed, I am afraid there will be *syphilis resurgens* again and again, invariably.

Yours, etc.,

CHARLES ENGEL.
22 Ocean Avenue,
Edgecliff,
New South Wales.
October 25, 1949.

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Commonwealth Bureau of Census and Statistics, Canberra (1946): Official Statistics, Commonwealth of Australia, Demographic, page 103.
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"BRITISH MEDICAL JOURNAL": REDUCED SUBSCRIPTION FOR MEDICAL STUDENTS.

SIR: Some weeks ago you published advice of the kind offer of the Editor of the *British Medical Journal* to extend to all medical students who have begun their clinical studies the opportunity of subscribing to that journal at the reduced rate of 10s. 6d. (sterling) *per annum*.

Numerous requests have been received in this office, and it would be appreciated if you would insert a note to the effect that supplies of application forms have been received and are now available from the Deans of the medical schools.

Yours, etc.,

J. H. PRIESTLEY,
Assistant General Secretary.
Federal Council of the British Medical Association in Australia,
135 Macquarie Street,
Sydney.
October 26, 1949.

Special Correspondence.

NEW ZEALAND LETTER.

FROM OUR SPECIAL CORRESPONDENT.

OCTOBER, 1949, has seen the passing of two Amendment Acts of some importance relating to medicine under Social Security. Both were hurried through in the last weeks of the session, and aroused little discussion. Members of Parliament have all eyes and ears on the general election to be held at the end of November, 1949. On the outcome of this there is little confident prediction. The Labour Party has held power in this Parliament by a very thin margin, and its handling of the conscription issue, waterfront disputes, and other questions has caused much discussion.

The Medical Practitioners Amendment Act 1949.

The Medical Practitioners Amendment Act 1949 deals with disciplinary matters in medical practice under social security. In the eight years since general medical services were begun, there has been no mechanism for dealing with errors and abuses. The present Act remedies this position by constituting a Medical Practitioners' Disciplinary Committee and Divisional Disciplinary Committees (of six members in the case of the four main centres, and three in smaller centres).

That the responsibility for discipline has been entrusted to the medical profession is shown by the facts that all members of all the committees are medical men, and in all cases are appointed by the Council of the New Zealand Branch of the British Medical Association, except one member of the five constituting the Central Committee, who is to be appointed by the Minister of Health.

The Disciplinary Committees are to deal with all charges against practitioners—anyone may make the charges—which

fall short of grave impropriety or infamous conduct. In the latter case, the existing Medical Council remains the appropriate body, and it is also now constituted a "higher court" to which appeals from the Disciplinary Committees go. Above the Medical Council remains, as at present, the Supreme Court. The Disciplinary Committee may inflict fines of up to £100, censure a defendant, or order costs of an inquiry to be paid. The fines, queerly, are to be paid to the British Medical Association, but this appears to be part of a plan to secure maintenance funds for the conduct of these inquiries, though Government responsibility for such funds is in part acknowledged. The Committees are to regulate their own procedure subject to any rules made by the Governor-General by Order in Council

The Medical Council remains much as before. It may now inflict fines of up to £100 (£50 formerly) and it may move the Supreme Court to remove a name from the register as before.

These provisions are necessary and long overdue, and on the whole have been fairly planned. How far they will be able to cope with the admitted abuses of a scheme which is in some of its essential features prone to error, remains to be seen.

The Social Security Amendment Act 1949.

Part One of *The Social Security Amendment Act* 1949 deals with the rates of the following monetary benefits: Superannuation, Age Benefits, Widows', Orphans', Invalids', Sickness, Unemployment, and Miners'. In general these are all jacked up by about 10% from June 1, 1949.

Part Three deals with medical services, and is to come into force on a day to be appointed by the Minister of Health. The chief provisions are: (i) There is to be a prohibition of the simultaneous practice under capitation and fee-for-service, as occurs in a few cases at present. (ii) the Refund System hitherto approved by the British Medical Association is not to apply unless specially authorized by the British Medical Association after consultation with the Minister. (iii) Specialist services; no details are given, but power is taken to establish these under regulations with a recognized scale of fees, and a list of recognized specialists and provision for determining what constitutes a special service. (iv) The right of medical practitioners to recover fees at law is restored. The Disciplinary Committees may be used as referees by the Courts in such disputes.

It will thus be seen that some attempt has been made to implement the recommendations of the Joint Committee on Medical Services (see THE MEDICAL JOURNAL OF AUSTRALIA, October 9, 1948). The major issue, however, that of specialist services, is left to regulations.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

COURSE IN PÆDIATRICS.

THE Post-Graduate Committee in Medicine in the University of Sydney will conduct a course in pædiatrics at the Royal Alexandra Hospital for Children for one week beginning December 5, 1949. The programme is as follows:

Monday, December 5: 9.30 a.m., "Common Skin Diseases of Childhood", Dr. Henry Sharp; 10.45 a.m., "Intrathoracic Tuberculosis", Dr. W. P. MacCallum; 11.45 a.m., "Appendicitis", Dr. E. S. Stuckey; 2 p.m., symposium on "Poliomyelitis": "Epidemiology and Diagnosis", Dr. Lorimer Dods and Dr. Stephen Williams; "Treatment", Dr. Laurence Macdonald, assisted by Miss J. Ferguson.

Tuesday, December 6: 9.30 a.m., "Surgery of the Newly Born", Dr. T. Y. Nelson;; 10.45 a.m., ward rounds; 11.45 a.m., "Convulsive Disorders of Childhood", Dr. D. G. Hamilton; 2 p.m., "Therapeutics", Dr. Lorimer Dods; 3.15 p.m., "Minor Surgical Problems", Dr. F. N. Street .

Wednesday, December 7: 9.30 a.m., "Burns", Dr. J. Steigrad; 10.45 a.m., ward rounds; 11.45 a.m., "Care of the

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED OCTOBER 22, 1949.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.[2]	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.	Australia.[3]
Ankylostomiasis	*	1	..	..	*	..	..	..	1
Anthrax	*	..	..	..	..	..	..	..	..
Beriberi	*	*	*	*	*	*	..	*	..
Bilharziasis	*	..	..	..	..	..	..	..	..
Cerebro-spinal Meningitis	3(2)	..	..	..	..	1	..	..	4
Cholera	..	..	..	..	..	..	..	..	..
Coastal Fever(*a*)	*	*	*	*	*	*	*	*	..
Dengue	*	*	*	*	*	*	..	*	..
Diarrhœa (Infantile)	..	..	5(3)	..	..	..	..	..	5
Diphtheria	13(9)	..	1(1)	..	2(2)	..	..	..	16
Dysentery(*b*)	*	..	3(1)	..	2	..	..	..	5
Encephalitis Lethargica	..	..	..	..	..	..	..	..	..
Erysipelas	*	*	*	..	*	*	..	..	..
Filariasis	*	..	..	..	..	..	..	..	..
Helminthiasis	*	..	*	*	*	*	*	*	..
Hydatid	*	..	*	*	..	*	*	..	..
Influenza	*	*	*	..	*	*	..	*	..
Leprosy	..	..	..	..	..	..	..	..	..
Malaria(*c*)	*	(*c*)	(*c*)	(*c*)	(*c*)	(*c*)	(*c*)	(*c*)	(*c*)
Measles	*	*	*	..	*	*	..	1	1
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	7(2)	20(10)	..	..	1(1)	2(1)	..	..	30
Psittacosis	*	..	*	..	*	*	*	*	..
Puerperal Fever	..	..	1	..	..	..	..	..	1
Rubella(*h*)	*	*	9(5)	*	3(1)	*	..	2	14
Scarlet Fever	31(20)	18(8)	1	..	1(1)	3(2)	..	5	59
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	*	..	*	*	1(1)	*	*	*	1
Trachoma	*	*	*	*	*	*	*	*	..
Tuberculosis(*d*)	16(12)	16(12)	5(4)	..	7(5)	..	..	..	46
Typhoid Fever(*e*)	..	1	..	..	..	..	..	..	1
Typhus (Endemic)(*f*)	..	..	2	..	..	..	..	..	2
Undulant Fever	..	1(1)	..	..	..	..	..	..	1
Weil's Disease(*g*)	*	*	*	*	*	*	*	*	..
Whooping Cough	*	*	*	..	*	*	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] The form of this table is taken from the *Official Year Book of the Commonwealth of Australia*, Number 36, 1944–1945. Figures in parentheses are those for the metropolitan area.
[2] Figures not available.
[3] Figures incomplete owing to absence of returns from Northern Territory and South Australia.
* Not notifiable.
(*a*) Includes "Mossman" and "Sarina" fevers. (*b*) Includes amœbic and bacillary. (*c*) Statistics inexact with varying practice with regard to relapses in Service cases infected overseas. (*d*) Includes all forms except in Northern Territory, where only pulmonary tuberculosis is notifiable. (*e*) Includes enteric fever, paratyphoid fevers and other Salmonella infections. (*f*) Cases reported include scrub, murine and tick typhus. (*g*) Includes leptospiroses, Weil's and para-Weil's disease. (*h*) Notifiable disease in Queensland in females aged over fourteen years.

Premature Infant", Dr. S. E. L. Stening; 2 p.m., "Immunization", Dr. Stephen Williams; 3.15 p.m., demonstration of practical procedures, Dr. Judith Dey; 3.45 p.m., demonstration of nursing procedures, Sister E. M. Worth.

Thursday, December 8: 9.30 a.m., "Common Orthopædic Problems", Dr. Laurence Macdonald; 10.45 a.m., ward rounds; 11.45 a.m., "Treatment of Allergic Conditions in Childhood", Dr. S. E. L. Stening; 2 p.m., symposium on "Bronchiectasis in Childhood": "Medical Aspects", Dr. D. G. R. Vickery; "Bronchograms and Bronchoscopic Suction", Dr. Norman H. Meacle; "Methods of Administration of Aerosol Penicillin", Dr. A. Distin Morgan; "Surgical Aspects", Dr. T. Y. Nelson.

Friday, December 9: 9.30 a.m., "Renal Tract Infections", Dr. J. W. S. Laidley; 10.45 a.m., ward rounds; 11.45 a.m., "Plastic Surgery", Dr. David Dey; 2 p.m., question time; 3.15 p.m., symposium on "Congenital Heart Disease", Dr. S. G. Bradfield, Dr. Kerrod Voss and Dr. T. Y. Nelson.

An exhibition of pathological specimens illustrating bronchiectasis and congenital heart disease will be set up in the lecture theatre of the hospital by the Director of Pathology, Dr. Douglas Reye. Fee for attendance at the course is £3 3s., and those wishing to enrol are requested to communicate with the Course Secretary of the Committee, 131 Macquarie Street, Sydney, at an early date. Telephones: BU 5238-BW 7483.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE following additional qualifications have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1939, of New South Wales:

Bolliger, Walter, 300 Sailors Bay Road, Northbridge (M.B., B.S., 1945, D.P.H., 1947, Univ. Sydney, D.T.M. and H., 1949, Univ. Liverpool), Dip. Ind. Health, Univ. Edinburgh, 1949.

O'Brien, John Patrick Edward, Prince Henry Hospital, Little Bay (M.B., B.S., 1938, Univ. Sydney), M.D., 1949, Univ. Sydney.

TASMANIA.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners:

Lee, Harrie Bertie, M.B., B.S., 1911 (Univ. Melbourne), Hobart.

Thomson, Harold Roberts, M.B., B.S., 1942 (Univ. Melbourne), Launceston.

Obituary.

RUPERT GEORGE ARNOTT.

WE regret to announce the death of Dr. Rupert George Arnott, which occurred on October 29, 1949, at Vaucluse, New South Wales.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Fowler, Frank William, M.B., B.S., 1947 (Univ. Sydney), 132 Glebe Road, Glebe.

Smith, Thomas Bruce, M.B., B.S., 1947 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.

Dwyer, Brian Eric, M.B., B.S., 1948 (Univ. Sydney), 2A St. Elmo Street, Mosman.

Strang, Errol James, M.B., B.S., 1948 (Univ. Sydney), 41 Hay Street, Croydon.

Desgrand, Vincent Geoffrey Scriven, M.B., 1942 (Univ. Sydney), Laurel Avenue, Turramurra.

Maitland, Herbert Lethington Chisholm, M.B., Ch.M., 1923 (Univ. Sydney), F.R.C.S., 1947 (England), 193 George Street, Bathurst, New South Wales.

Medical Appointments.

Dr. J. J. Elphinstone has been appointed Quarantine Officer, Onslow, Western Australia, under the *Quarantine Act*, 1908-1947.

Dr. D. J. Snow has been appointed Quarantine Officer, Wyndham, Western Australia, under the *Quarantine Act*, 1908-1947.

Diary for the Month.

Nov. 14.—Victorian Branch, B.M.A.: Finance, House and Library Subcommittee.
Nov. 15.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Nov. 16.—Western Australian Branch, B.M.A.: General Meeting.
Nov. 17.—New South Wales Branch, B.M.A.: Clinical Meeting.
Nov. 17.—Victorian Branch, B.M.A.: Executive Meeting.
Nov. 22.—New South Wales Branch, B.M.A.: Ethics Committee.
Nov. 23.—Victorian Branch, B.M.A.: Council Meeting.
Nov. 24.—South Australian Branch, B.M.A.: Clinical Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135 Macquarie Street, Sydney): Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales (Telephones: MW 2651-2.)

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